STIC Search Report
Biotech-Chem Library

STIC Database Tracking Number: 172748

TO: Ralph J Gitomer
Location: REM-3C18
Art Unit: 1655
Friday, December 16, 2005
Case Serial Number: 10/798986

From: Barb O'Bryen
Location: Biotech-Chem Library
Remsen 1a69
Phone: 571-272-2518

barbara.obryen@uspto.gov

Search Notes

=> fil capl; d que 15; d que 13; fil medl; d que 195; fil embase; d que 1115; d que 1116
FILE 'CAPLUS' ENTERED AT 16:04:57 ON 16 DEC 2005
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FILE COVERS 1907 - 16 Dec 2005 VOL 143 ISS 26
FILE LAST UPDATED: 15 Dec 2005 (20051215/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
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'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE

L1	493	SEA	FILE=CAPLUS	ABB=ON	CHIN W?/AU
L2	2192	SEA	FILE=CAPLUS	ABB=ON	KWON S?/AU
L4	16036	SEA	FILE=CAPLUS	ABB=ON	BIOSENSORS/CT
L5	2	SEA	FILE=CAPLUS	ABB=ON	(L1 OR L2) AND L4

Inventor search

L1	493	SEA	FILE=CAPLUS	ABB=ON	CHIN W?/AU
L2	2192	SEA	FILE=CAPLUS	ABB=ON	KWON S?/AU
L3	0	SEA	FILE=CAPLUS	ABB=ON	L1 AND L2

FILE 'MEDLINE' ENTERED AT 16:04:57 ON 16 DEC 2005

FILE LAST UPDATED: 15 DEC 2005 (20051215/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 will soon be available. For details on the 2005 reload, enter HELP RLOAD at an arrow promt (=>). See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

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This file contains CAS Registry Numbers for easy and accurate

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L77        447 SEA FILE=MEDLINE ABB=ON  CHIN W?/AU
L78        540 SEA FILE=MEDLINE ABB=ON  KWON S?/AU
L80       3896 SEA FILE=MEDLINE ABB=ON  BIOLOGICAL WARFARE+NT/CT
L81        716 SEA FILE=MEDLINE ABB=ON  CHEMICAL WARFARE+NT/CT
L82     102328 SEA FILE=MEDLINE ABB=ON  ENVIRONMENTAL EXPOSURE+NT/CT
L83      41512 SEA FILE=MEDLINE ABB=ON  AIR POLLUTION+NT/CT
L84     176076 SEA FILE=MEDLINE ABB=ON  EPITHELIAL CELLS+NT/CT
L85     174367 SEA FILE=MEDLINE ABB=ON  EPITHELIUM+NT/CT
L86     269379 SEA FILE=MEDLINE ABB=ON  RESPIRATORY SYSTEM+NT/CT
L87       7060 SEA FILE=MEDLINE ABB=ON  CILIA/CT
L88        485 SEA FILE=MEDLINE ABB=ON  GOBLET CELLS+NT/CT
L89      10594 SEA FILE=MEDLINE ABB=ON  MUCINS+NT/CT
L90       8627 SEA FILE=MEDLINE ABB=ON  BIOSENSING TECHNIQUES+NT/CT
L93     758406 SEA FILE=MEDLINE ABB=ON  CELLS, CULTURED+NT/CT
L95          1 SEA FILE=MEDLINE ABB=ON  (L77 OR L78) AND (L80 OR L81 OR L82
               OR L83) AND ((L84 OR L85 OR L86 OR L87 OR L88 OR L89 OR L90)
               OR L93)
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FILE 'EMBASE' ENTERED AT 16:04:57 ON 16 DEC 2005

FILE COVERS 1974 TO 15 Dec 2005 (20051215/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L102       395 SEA FILE=EMBASE ABB=ON  CHIN W?/AU
L103       476 SEA FILE=EMBASE ABB=ON  KWON S?/AU
L115         0 SEA FILE=EMBASE ABB=ON  L102 AND L103
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L102       395 SEA FILE=EMBASE ABB=ON  CHIN W?/AU
L103       476 SEA FILE=EMBASE ABB=ON  KWON S?/AU
L104      3037 SEA FILE=EMBASE ABB=ON  BIOLOGICAL WARFARE/CT
L105       996 SEA FILE=EMBASE ABB=ON  CHEMICAL WARFARE/CT
L106     22836 SEA FILE=EMBASE ABB=ON  ENVIRONMENTAL EXPOSURE/CT
L107     84207 SEA FILE=EMBASE ABB=ON  "AIR AND AIR RELATED PHENOMENA"+NT/CT
L108      3900 SEA FILE=EMBASE ABB=ON  RESPIRATORY EPITHELIUM/CT
L109    187739 SEA FILE=EMBASE ABB=ON  CELL CULTURE/CT
L110       218 SEA FILE=EMBASE ABB=ON  EUKARYOTIC FLAGELLUM/CT
L111      2385 SEA FILE=EMBASE ABB=ON  GOBLET CELL/CT
L112      6530 SEA FILE=EMBASE ABB=ON  MUCIN/CT
L113      7849 SEA FILE=EMBASE ABB=ON  BIOSENSOR+NT/CT
L114     15116 SEA FILE=EMBASE ABB=ON  BIOASSAY/CT
L116         0 SEA FILE=EMBASE ABB=ON  (L102 OR L103) AND (L104 OR L105 OR
               L106 OR L107) AND (L108 OR L109 OR L110 OR L111 OR L112 OR
               L113 OR L114)
```

=> fil jic pascal caba biotechno esbio ntis nioshtic enviroeng healsafe inspec biosis confsci lifesci polluab toxcenter ceaba wpix scisearch

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FILE 'JICST-EPLUS' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'PASCAL' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'CABA' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'BIOTECHNO' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'ESBIOBASE' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'NTIS' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'NIOSHTIC' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'ENVIROENG' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'HEALSAFE' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'INSPEC' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'BIOSIS' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'CONFSCI' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'LIFESCI' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'POLLUAB' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'TOXCENTER' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'CEABA-VTB' ENTERED AT 16:05:30 ON 16 DEC 2005

FILE 'WPIX' ENTERED AT 16:05:30 ON 16 DEC 2005

Searched by Barb O'Bryen, STIC 2-2518

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FILE 'SCISEARCH' ENTERED AT 16:05:30 ON 16 DEC 2005
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=> d que l62; d que l63
L1          493 SEA FILE=CAPLUS ABB=ON  CHIN W?/AU
L2         2192 SEA FILE=CAPLUS ABB=ON  KWON S?/AU
L50        3121 SEA L1
L51        7974 SEA L2
L62           2 SEA L50 AND L51



L1          493 SEA FILE=CAPLUS ABB=ON  CHIN W?/AU
L2         2192 SEA FILE=CAPLUS ABB=ON  KWON S?/AU
L50        3121 SEA L1
L51        7974 SEA L2
L52     2197473 SEA BIOSENSOR# OR SENSOR#
L53      290424 SEA BIOASSAY?
L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L56      955407 SEA (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR QUALITY)
L57      158030 SEA AIRBORNE OR AIR BORNE
L58       17285 SEA (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L63           0 SEA (L50 OR L51) AND (L52 OR L53) AND (L54 OR (L56 OR L57 OR
                L58))


=> dup rem l95,l5,l62
FILE 'MEDLINE' ENTERED AT 16:05:42 ON 16 DEC 2005

FILE 'CAPLUS' ENTERED AT 16:05:42 ON 16 DEC 2005
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FILE 'BIOSIS' ENTERED AT 16:05:42 ON 16 DEC 2005
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FILE 'SCISEARCH' ENTERED AT 16:05:42 ON 16 DEC 2005
Copyright (c) 2005 The Thomson Corporation
PROCESSING COMPLETED FOR L95
PROCESSING COMPLETED FOR L5
PROCESSING COMPLETED FOR L62
L127           5 DUP REM L95 L5 L62 (0 DUPLICATES REMOVED)
                 ANSWER '1' FROM FILE MEDLINE
                 ANSWERS '2-3' FROM FILE CAPLUS
                 ANSWER '4' FROM FILE BIOSIS
                 ANSWER '5' FROM FILE SCISEARCH

=> d iall 1; d ibib ed abs hitind 2-3; d iall 4-5

L127 ANSWER 1 OF 5     MEDLINE on STN
ACCESSION NUMBER:    2004189161      MEDLINE
DOCUMENT NUMBER:     PubMed ID: 14986108
TITLE:               Molecular cloning of a cytosolic ascorbate peroxidase cDNA
                     from cell cultures of sweet potato and its expression in
                     response to stress.
AUTHOR:              Park S-Y; Ryu S-H; Jang I-C; Kwon S-Y; Kim J-G;
                     Kwak S-S
CORPORATE SOURCE:    Laboratory of Environmental Biotechnology, Korea Research
                     Institute of Bioscience and Biotechnology, Oun-dong 52,
```

Yusong-gu, 305-806 Daejeon, Korea.
SOURCE: Molecular genetics and genomics : MGG, (2004 Apr) 271 (3) 339-46. Electronic Publication: 2004-02-17.
Journal code: 101093320. ISSN: 1617-4615.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AY206407
ENTRY MONTH: 200405
ENTRY DATE: Entered STN: 20040416
Last Updated on STN: 20040529
Entered Medline: 20040528

ABSTRACT:
A cDNA encoding a cytosolic ascorbate peroxidase (APX), swAPX1, was isolated from cell cultures of sweet potato (Ipomoea batatas) by cDNA library screening, and its expression in the context of various environmental stresses was investigated. swAPX1 contains an ORF of 250 amino acids (27.5 kDa) encoding a protein with a pI value of 5.32. The swAPX1 ORF does not code for a transit peptide, suggesting that the product is a cytosolic isoform. RNA blot analysis showed that swAPX1 gene is expressed in cultured cells and mature leaves, but not in stems, non-storage or storage roots of sweet potato. The level of swAPX1 RNA progressively increased during cell growth in suspension cultures. In leaf tissues, the gene responded differentially to various abiotic stresses, as revealed by RT-PCR analysis. swAPX1 was highly induced in leaves by wounding, and treatment with methyl viologen (50 microM), hydrogen peroxide (440 mM), abscisic acid (ABA; 100 microM) or exposure to high temperature (37 degrees C). In addition, the gene was strongly induced in the leaves following inoculation with a bacterial pathogen (Pectobacterium chrysanthemi). These results indicate that swAPX1 may be involved in hydrogen peroxide-detoxification and thus help to overcome the oxidative stress induced by abiotic and biotic stresses.

CONTROLLED TERM:
Abscisic Acid: PD, pharmacology
Amino Acid Sequence
Blotting, Northern
Cell Division
Cells, Cultured
Cloning, Molecular
Cytosol
*DNA, Complementary: GE, genetics
Environmental Exposure
*Gene Expression Regulation, Enzymologic
Gene Expression Regulation, Plant
Gene Library
Herbicides: PD, pharmacology
Hydrogen Peroxide: PD, pharmacology
*Ipomoea batatas: EN, enzymology
Ipomoea batatas: GE, genetics
Molecular Sequence Data
Oxidants: PD, pharmacology
*Oxidative Stress
Paraquat: PD, pharmacology
*Peroxidases: GE, genetics
Peroxidases: ME, metabolism
Plant Growth Regulators: PD, pharmacology
Plant Leaves: CY, cytology
*Plant Leaves: ME, metabolism
RNA, Plant: GE, genetics
RNA, Plant: ME, metabolism
Research Support, Non-U.S. Gov't

Reverse Transcriptase Polymerase Chain Reaction
Sequence Homology, Amino Acid
Temperature
Wounds and Injuries

CAS REGISTRY NO.: 21293-29-8 (Abscisic Acid); 4685-14-7 (Paraquat); 7722-84-1 (Hydrogen Peroxide)

CHEMICAL NAME: 0 (DNA, Complementary); 0 (Herbicides); 0 (Oxidants); 0 (Plant Growth Regulators); 0 (RNA, Plant); EC 1.11.1. (Peroxidases); EC 1.11.1.11 (ascorbate peroxidase)

L127 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:936410 CAPLUS
DOCUMENT NUMBER: 142:351522
TITLE: Electrical detection of kinase assay using multi walled-carbon nanotube (MWCNT) nanoelectrode
AUTHOR(S): Lee, Jae Shin; Kim, Do Hyun; Lee, Seok Jae; Park, Jong Pil; Park, Tae Jung; Lee, Sang Yup; Jung, Dae-Hwan; Jung, Hee-Tae; Kim, Jin Hee; **Kwon, Seong Ku**
CORPORATE SOURCE: Dept. of Chemical and Biomolecular engineering, and Center for Ultramicrochemical Process Systems, KAIST, Yuseong-gu, Daejeon, 305-701, S. Korea
SOURCE: Special Publication - Royal Society of Chemistry (2004), 297(Micro Total Analysis Systems 2004, Volume 2), 124-126
CODEN: SROCDO; ISSN: 0260-6291
PUBLISHER: Royal Society of Chemistry
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 06 Nov 2004
AB We have demonstrated the use of MWCNT as a nanoscale probe to monitor the activity of enzyme kinase. To immobilize the substrate peptide using carbodiimide chemical, plasma or strong acid treatments were used to induce carboxyl groups on the sidewall of MWCNTs. After the substrate peptide immobilization, increase of conductance from MWCNT devices was observed When peptide modified MWCNTs react with enzyme kinase, conductance decreases by several orders of magnitude, and this conductance change can be explained by the phosphorylation reaction of enzyme kinase. When the sample was incubated with phosphatase to dephosphorylate the substrate peptide, nearly complete recovery of the conductance signal has been observed and we can confirm that we have monitored the kinase activity.
CC 9-7 (Biochemical Methods)
IT **Biosensors**
Blood plasma
Carboxyl group
Electric conductivity
Immobilization, molecular or cellular
(elec. detection of kinase assay using multi walled-carbon nanotube(MWCNT) nanoelectrode)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L127 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:224372 CAPLUS
DOCUMENT NUMBER: 134:234036
TITLE: Non- or minimally-invasive monitoring methods using particle delivery methods
INVENTOR(S): **Kwon, Sung-yun**

PATENT ASSIGNEE(S): Powderject Research Limited, UK
SOURCE: U.S., 10 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6207400	B1	20010327	US 1999-390068	19990903
JP 2002524120	T2	20020806	JP 2000-568385	19990903
US 2001029957	A1	20011018	US 2001-803828	20010312
US 6482604	B2	20021119		
US 2002102625	A1	20020801	US 2001-22633	20011217
US 6602678	B2	20030805		
US 2005064528	A1	20050324	US 2004-499061	20041105

PRIORITY APPLN. INFO.:

US 1998-99157P	P	19980904
US 1999-390068	A1	19990903
WO 1999-GB2914	W	19990903
US 2001-803828	A2	20010312
US 2001-22633	A1	20011217
WO 2002-US37604	W	20021213

ED Entered STN: 29 Mar 2001
AB Methods for sampling an analyte present in a biol. system are provided. The methods entail use of particle delivery methods to obtain a sample of an analyte of interest from the system. Using a needleless syringe particle delivery device, lactose powder was delivered to the skin of diabetic patients. A LifeScan glucose detection membrane strip was moistened with hydroel and applied to the powder-injected site for 1 min. The color intensity of the membranes were quantified with a Bio-Rad densitometer. The interstitial glucose levels were compared with blood glucose concns.
IC ICM C12Q001-54
ICS C12Q001-00; C12Q001-26; C12M001-00
INCL 435014000
CC 9-16 (Biochemical Methods)
Section cross-reference(s): 14
IT Absorbents
Bioassay
Biochemical molecules
Biosensors
Blood analysis
Body fluid
Diabetes mellitus
Hydrogels
Mucous membrane
Particles
Permeability
Sampling
Skin
(non- or minimally-invasive monitoring methods using particle delivery methods)
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L127 ANSWER 4 OF 5 BIOSIS

ACCESSION NUMBER: 2004:286506 BIOSIS
DOCUMENT NUMBER: PREV200400285263
TITLE: Premature Activation Mechanisms of Trypsin.
AUTHOR(S): Yang, Kai [Reprint Author]; Ding, Yongxue; **Kwon, Soonjo; Chin, Wei-Chun**
CORPORATE SOURCE: The Department of Biological Science, Florida State University, Biology Unit One, Tallahassee, FL, 32306, USA
kai@bio.fsu.edu
SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 459.23. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

ABSTRACT:Under normal physiological conditions, trypsin remains inactive as trypsinogen inside the pancreas. Upon entering the small intestine, trypsinogen is converted to active trypsin. Acute pancreatitis is thought to begin with the premature activation of trypsinogen in the pancreas. However, the exact initiating mechanisms of this premature activation are still not clear. In this study we isolated the zymogen granules (ZGs) from pancreatic acinar cells. The pH fluctuations ((pH)G), Ca2+ changes ((Ca2+)G) and trypsin premature activation inside ZGs were simultaneously monitored with fluorescent probes. We also investigated possible pharmacological inhibitors and blockers for this premature activation process. Our results showed that a sustained increase of intracellular Ca2+ could trigger K+ influx into ZGs through Ca2+-activated K+ channels. The influx of K+ can mobilize bound Ca2+ by K+/Ca2+ ion-exchange to increase (Ca2+)G and bound H+ by K+/H+ ion-exchange to decrease (pH)G. Both the increase of (Ca2+)G and the decrease of (pH)G can facilitate trypsinogen autoactivation and stabilize trypsin activity. Our investigations also showed that trypsin premature activation can be activated by (Ca2+) above 300 nM and blocked by apamin (100 muM) and TEA (20 mM). This research project was supported by FSU Cornerstone Program and ABMRF.

CONCEPT CODE:
General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Enzymes - General and comparative studies: coenzymes 10802
Digestive system - Physiology and biochemistry 14004
Digestive system - Pathology 14006
Endocrine - General 17002
Endocrine - Pancreas 17008

INDEX TERMS: Major Concepts
Digestive System (Ingestion and Assimilation); Enzymology (Biochemistry and Molecular Biophysics)

INDEX TERMS: Parts, Structures, & Systems of Organisms
pancreas: digestive system, endocrine system; pancreatic acinar cell: endocrine system, zymogen granules; small intestine: digestive system

INDEX TERMS: Diseases
acute pancreatitis: digestive system disease
Pancreatitis (MeSH)

INDEX TERMS: Chemicals & Biochemicals
trypsin [EC 3.4.21.4]: premature activation mechanism

INDEX TERMS: Miscellaneous Descriptors
potassium fluoride/calcium ion exchange

Searched by Barb O'Bryen, STIC 2-2518

REGISTRY NUMBER: 9002-07-7 (trypsin)
9002-07-7 (EC 3.4.21.4)

L127 ANSWER 5 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:619852 SCISEARCH
THE GENUINE ARTICLE: 806ZA
TITLE: Premature activation mechanisms of trypsin
AUTHOR: Yang K (Reprint); Ding Y X; **Kwon S**; **Chin W C**
CORPORATE SOURCE: Florida State Univ, Dept Biol Sci, Biol Unit 1, Tallahassee, FL 32306 USA; Florida State Univ, Dept Chem Engn, Tallahassee, FL 32306 USA; Florida State Univ, Biomed Engn Program, Tallahassee, FL 32306 USA
COUNTRY OF AUTHOR: USA
SOURCE: FASEB JOURNAL, (23 MAR 2004) Vol. 18, No. 4, Supp. [S], pp. A705-A705.
ISSN: 0892-6638.
PUBLISHER: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
ENTRY DATE: Entered STN: 29 Jul 2004
Last Updated on STN: 29 Jul 2004
CATEGORY: BIOCHEMISTRY & MOLECULAR BIOLOGY; BIOLOGY; CELL BIOLOGY

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=> fil embase; d que l118; d que l123; d que l126

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 FILE COVERS 1974 TO 15 Dec 2005 (20051215/ED)

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 substance identification.


L104        3037 SEA FILE=EMBASE ABB=ON  BIOLOGICAL WARFARE/CT
L105         996 SEA FILE=EMBASE ABB=ON  CHEMICAL WARFARE/CT
L106       22836 SEA FILE=EMBASE ABB=ON  ENVIRONMENTAL EXPOSURE/CT
L107       84207 SEA FILE=EMBASE ABB=ON  "AIR AND AIR RELATED PHENOMENA"+NT/CT
L108        3900 SEA FILE=EMBASE ABB=ON  RESPIRATORY EPITHELIUM/CT
L113        7849 SEA FILE=EMBASE ABB=ON  BIOSENSOR+NT/CT
L114       15116 SEA FILE=EMBASE ABB=ON  BIOASSAY/CT
L118           1 SEA FILE=EMBASE ABB=ON  (L104 OR L105 OR L106 OR L107) AND
                 L108 AND (L113 OR L114)


L104        3037 SEA FILE=EMBASE ABB=ON  BIOLOGICAL WARFARE/CT
L105         996 SEA FILE=EMBASE ABB=ON  CHEMICAL WARFARE/CT
L106       22836 SEA FILE=EMBASE ABB=ON  ENVIRONMENTAL EXPOSURE/CT
L107       84207 SEA FILE=EMBASE ABB=ON  "AIR AND AIR RELATED PHENOMENA"+NT/CT
L108        3900 SEA FILE=EMBASE ABB=ON  RESPIRATORY EPITHELIUM/CT
L109      187739 SEA FILE=EMBASE ABB=ON  CELL CULTURE/CT
L119      578914 SEA FILE=EMBASE ABB=ON  IN VITRO STUDY/CT
L121        4485 SEA FILE=EMBASE ABB=ON  MONOLAYER CULTURE/CT
L122        1551 SEA FILE=EMBASE ABB=ON  CILIARY MOTILITY/CT
L123           4 SEA FILE=EMBASE ABB=ON  (L104 OR L105 OR L106 OR L107) AND
                 L108 AND L109 AND (L119 OR (L121 OR L122))


L104        3037 SEA FILE=EMBASE ABB=ON  BIOLOGICAL WARFARE/CT
L105         996 SEA FILE=EMBASE ABB=ON  CHEMICAL WARFARE/CT
L106       22836 SEA FILE=EMBASE ABB=ON  ENVIRONMENTAL EXPOSURE/CT
L107       84207 SEA FILE=EMBASE ABB=ON  "AIR AND AIR RELATED PHENOMENA"+NT/CT
L108        3900 SEA FILE=EMBASE ABB=ON  RESPIRATORY EPITHELIUM/CT
L109      187739 SEA FILE=EMBASE ABB=ON  CELL CULTURE/CT
L117          12 SEA FILE=EMBASE ABB=ON  (L104 OR L105 OR L106 OR L107) AND
                 L108 AND L109
L125       38308 SEA FILE=EMBASE ABB=ON  QUANTITATIVE ANALYSIS/CT
L126           1 SEA FILE=EMBASE ABB=ON  L117 AND L125

=> s l118 or l123 or l126

L128           6 L118 OR L123 OR L126

=> fil capl; d que l7; d que l27; d que l28; d que l30; d que l37

FILE 'CAPLUS' ENTERED AT 16:08:36 ON 16 DEC 2005
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 16 Dec 2005 VOL 143 ISS 26
FILE LAST UPDATED: 15 Dec 2005 (20051215/ED)

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http://www.cas.org/infopolicy.html
'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE

L4	16036	SEA FILE=CAPLUS ABB=ON BIOSENSORS/CT
L6	3734	SEA FILE=CAPLUS ABB=ON EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR GOBLET?/OBI)
L7	1	SEA FILE=CAPLUS ABB=ON L4 AND L6

L6	3734	SEA FILE=CAPLUS ABB=ON EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR GOBLET?/OBI)
L8	208880	SEA FILE=CAPLUS ABB=ON CULTUR?/OBI
L9	244	SEA FILE=CAPLUS ABB=ON L6 AND L8
L10	42474	SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT
L11	147315	SEA FILE=CAPLUS ABB=ON AIR/OBI (L)POLLUTION/CW
L12	4705	SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI)(L)WARF ARE/OBI
L13	13214	SEA FILE=CAPLUS ABB=ON AIR/OBI(L)MONITOR?/OBI
L26	20819	SEA FILE=CAPLUS ABB=ON SAMPLING/CW
L27	4	SEA FILE=CAPLUS ABB=ON L9 AND ((L10 OR L11 OR L12 OR L13) OR L26)

L6	3734	SEA FILE=CAPLUS ABB=ON EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR GOBLET?/OBI)
L10	42474	SEA FILE=CAPLUS ABB=ON ENVIRONMENTAL POLLUTION/CT
L11	147315	SEA FILE=CAPLUS ABB=ON AIR/OBI (L)POLLUTION/CW
L12	4705	SEA FILE=CAPLUS ABB=ON (BIOLOGICAL/OBI OR CHEMICAL/OBI)(L)WARF ARE/OBI
L13	13214	SEA FILE=CAPLUS ABB=ON AIR/OBI(L)MONITOR?/OBI
L16	11248	SEA FILE=CAPLUS ABB=ON AIRBORNE PARTICLES/CT
L17	56518	SEA FILE=CAPLUS ABB=ON TOXICITY/CT
L18	4102	SEA FILE=CAPLUS ABB=ON ECOTOXICITY/CT
L19	9069	SEA FILE=CAPLUS ABB=ON BIOASSAY/CT
L22	14	SEA FILE=CAPLUS ABB=ON L6 AND L19
L26	20819	SEA FILE=CAPLUS ABB=ON SAMPLING/CW
L28	3	SEA FILE=CAPLUS ABB=ON L22 AND ((L10 OR L11 OR L12 OR L13) OR

L26 OR (L16 OR L17 OR L18))

L4	16036	SEA FILE=CAPLUS ABB=ON	BIOSENSORS/CT
L6	3734	SEA FILE=CAPLUS ABB=ON	EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR GOBLET?/OBI)
L7	1	SEA FILE=CAPLUS ABB=ON	L4 AND L6
L8	208880	SEA FILE=CAPLUS ABB=ON	CULTUR?/OBI
L9	244	SEA FILE=CAPLUS ABB=ON	L6 AND L8
L10	42474	SEA FILE=CAPLUS ABB=ON	ENVIRONMENTAL POLLUTION/CT
L11	147315	SEA FILE=CAPLUS ABB=ON	AIR/OBI (L)POLLUTION/CW
L12	4705	SEA FILE=CAPLUS ABB=ON	(BIOLOGICAL/OBI OR CHEMICAL/OBI)(L)WARFARE/OBI
L13	13214	SEA FILE=CAPLUS ABB=ON	AIR/OBI(L)MONITOR?/OBI
L16	11248	SEA FILE=CAPLUS ABB=ON	AIRBORNE PARTICLES/CT
L17	56518	SEA FILE=CAPLUS ABB=ON	TOXICITY/CT
L18	4102	SEA FILE=CAPLUS ABB=ON	ECOTOXICITY/CT
L19	9069	SEA FILE=CAPLUS ABB=ON	BIOASSAY/CT
L20	427058	SEA FILE=CAPLUS ABB=ON	59/SC,SX – Section code - Air pollution & industrial hygiene
L21	615834	SEA FILE=CAPLUS ABB=ON	4/SC,SX – Section code - Toxicology
L22	14	SEA FILE=CAPLUS ABB=ON	L6 AND L19
L25	74	SEA FILE=CAPLUS ABB=ON	L8 AND L20 AND L21 AND ((L10 OR L11 OR L12 OR L13) OR L16)
L26	20819	SEA FILE=CAPLUS ABB=ON	SAMPLING/CW
L27	4	SEA FILE=CAPLUS ABB=ON	L9 AND ((L10 OR L11 OR L12 OR L13) OR L26)
L28	3	SEA FILE=CAPLUS ABB=ON	L22 AND ((L10 OR L11 OR L12 OR L13) OR L26 OR (L16 OR L17 OR L18))
L29	3	SEA FILE=CAPLUS ABB=ON	L25 AND L26
L30	3	SEA FILE=CAPLUS ABB=ON	L29 NOT (L7 OR L27 OR L28)

L4	16036	SEA FILE=CAPLUS ABB=ON	BIOSENSORS/CT
L6	3734	SEA FILE=CAPLUS ABB=ON	EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR GOBLET?/OBI)
L10	42474	SEA FILE=CAPLUS ABB=ON	ENVIRONMENTAL POLLUTION/CT
L11	147315	SEA FILE=CAPLUS ABB=ON	AIR/OBI (L)POLLUTION/CW
L12	4705	SEA FILE=CAPLUS ABB=ON	(BIOLOGICAL/OBI OR CHEMICAL/OBI)(L)WARFARE/OBI
L13	13214	SEA FILE=CAPLUS ABB=ON	AIR/OBI(L)MONITOR?/OBI
L16	11248	SEA FILE=CAPLUS ABB=ON	AIRBORNE PARTICLES/CT
L17	56518	SEA FILE=CAPLUS ABB=ON	TOXICITY/CT
L18	4102	SEA FILE=CAPLUS ABB=ON	ECOTOXICITY/CT
L19	9069	SEA FILE=CAPLUS ABB=ON	BIOASSAY/CT
L20	427058	SEA FILE=CAPLUS ABB=ON	59/SC,SX
L21	615834	SEA FILE=CAPLUS ABB=ON	4/SC,SX
L26	20819	SEA FILE=CAPLUS ABB=ON	SAMPLING/CW
L31	647	SEA FILE=CAPLUS ABB=ON	(CILIA?(2A)BEAT?)/BI
L32	2600	SEA FILE=CAPLUS ABB=ON	(ELECTRICAL RESPONS?)/BI
L33	1144	SEA FILE=CAPLUS ABB=ON	.(SECRET?(2A)MUCIN#)/BI
L37	8	SEA FILE=CAPLUS ABB=ON	L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11 OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20) OR L26) AND L21

=> s (l7 or l27 or l28 or l30 or l37) not l5

L129 18 (L7 OR L27 OR L28 OR L30 OR L37) NOT (L5) previously printed w/ inventor search

```
=> fil medl; d que l101; s l101 not l95

FILE 'MEDLINE' ENTERED AT 16:08:38 ON 16 DEC 2005

 FILE LAST UPDATED: 15 DEC 2005 (20051215/UP). FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 will soon be available. For details
 on the 2005 reload, enter HELP RLOAD at an arrow promt (=>).
 See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

 OLDMEDLINE is covered back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2006 vocabulary.

 This file contains CAS Registry Numbers for easy and accurate

L80          3896 SEA FILE=MEDLINE ABB=ON  BIOLOGICAL WARFARE+NT/CT
L81           716 SEA FILE=MEDLINE ABB=ON  CHEMICAL WARFARE+NT/CT
L82        102328 SEA FILE=MEDLINE ABB=ON  ENVIRONMENTAL EXPOSURE+NT/CT
L83         41512 SEA FILE=MEDLINE ABB=ON  AIR POLLUTION+NT/CT
L84        176076 SEA FILE=MEDLINE ABB=ON  EPITHELIAL CELLS+NT/CT
L85        174367 SEA FILE=MEDLINE ABB=ON  EPITHELIUM+NT/CT
L86        269379 SEA FILE=MEDLINE ABB=ON  RESPIRATORY SYSTEM+NT/CT
L87          7060 SEA FILE=MEDLINE ABB=ON  CILIA/CT
L88           485 SEA FILE=MEDLINE ABB=ON  GOBLET CELLS+NT/CT
L89         10594 SEA FILE=MEDLINE ABB=ON  MUCINS+NT/CT
L93        758406 SEA FILE=MEDLINE ABB=ON  CELLS, CULTURED+NT/CT
L100        60664 SEA FILE=MEDLINE ABB=ON  AIR POLLUTANTS+NT/CT
L101            9 SEA FILE=MEDLINE ABB=ON  ((L80 OR L81 OR L82 OR L83) OR L100)
                  AND (L84 OR L85) AND L86 AND L93 AND (L87 OR L88 OR L89)


L130            9 L101 NOT L95 previously printed

=> fil jic pascal caba biotechno esbio ntis nioshtic enviroeng healsafe inspec
biosis confsci lifesci polluab toxcenter ceaba wpix scisearch
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```
=> d que l66; d que l67; d que l68; d que l73; d que l74
L52     2197473 SEA BIOSENSOR# OR SENSOR#
L53      290424 SEA BIOASSAY?
L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L55     3624943 SEA CULTUR?
L56      955407 SEA (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR QUALITY)
L57      158030 SEA AIRBORNE OR AIR BORNE
L58       17285 SEA (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L65         122 SEA (L52 OR L53) AND L54 AND L55
L66          16 SEA L65 AND (L56 OR L57 OR L58)
```

```
L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L55     3624943 SEA CULTUR?
L56      955407 SEA (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR QUALITY)
L57      158030 SEA AIRBORNE OR AIR BORNE
L58       17285 SEA (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L59        4285 SEA (CILIA?(2A) BEAT?)
L60        7049 SEA (ELECTRICAL RESPONS?)
L61        4997 SEA (SECRET?(2A) MUCIN#)
L67           2 SEA L54(5A) L55 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR
                L61)


L52     2197473 SEA BIOSENSOR# OR SENSOR#
L53      290424 SEA BIOASSAY?
L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L56      955407 SEA (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR QUALITY)
L57      158030 SEA AIRBORNE OR AIR BORNE
L58       17285 SEA (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L59        4285 SEA (CILIA?(2A) BEAT?)
L60        7049 SEA (ELECTRICAL RESPONS?)
L61        4997 SEA (SECRET?(2A) MUCIN#)
L64        1378 SEA (L52 OR L53) AND L54
L68           1 SEA L64 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR L61)


L52     2197473 SEA BIOSENSOR# OR SENSOR#
L53      290424 SEA BIOASSAY?
L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L59        4285 SEA (CILIA?(2A) BEAT?)
L60        7049 SEA (ELECTRICAL RESPONS?)
L61        4997 SEA (SECRET?(2A) MUCIN#)
L64        1378 SEA (L52 OR L53) AND L54
L69          32 SEA L64 AND (L59 OR L60 OR L61)
L71     1500078 SEA TOXICITY/CT OR PHARMACOLOGICAL OR SCREEN?/TI
L72     4696113 SEA SAMPL?
L73           9 SEA L69 AND (L71 OR L72)


L54       29232 SEA EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L55     3624943 SEA CULTUR?
L56      955407 SEA (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR QUALITY)
L57      158030 SEA AIRBORNE OR AIR BORNE
L58       17285 SEA (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L72     4696113 SEA SAMPL?
L74           6 SEA L54(5A) L55 AND L72 AND (L56 OR L57 OR L58)

=> s l66-168
L131         19 (L66 OR L67 OR L68)

=> s l73-174
L132         15 (L73 OR L74)
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=> s l131-l132
L135        34 (L131 OR L132)


=> dup rem l130,l129,l128,l135
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PROCESSING COMPLETED FOR L130
PROCESSING COMPLETED FOR L129
PROCESSING COMPLETED FOR L128
PROCESSING COMPLETED FOR L135
L136         47 DUP REM L130 L129 L128 L135 (20 DUPLICATES REMOVED)
                ANSWERS '1-9' FROM FILE MEDLINE
                ANSWERS '10-26' FROM FILE CAPLUS
                ANSWERS '27-31' FROM FILE EMBASE
                ANSWERS '32-33' FROM FILE PASCAL
                ANSWERS '34-36' FROM FILE ESBIOBASE
                ANSWER '37' FROM FILE NIOSHTIC
                ANSWER '38' FROM FILE INSPEC
                ANSWERS '39-43' FROM FILE BIOSIS
                ANSWERS '44-47' FROM FILE TOXCENTER

=> d iall 1-9; d ibib ed abs hitind 10-26; d iall 27-47; fil hom
```

Gene Expression: DE, drug effects
Guinea Pigs
*Industrial Waste
Metals: TO, toxicity
Mucins: BI, biosynthesis
Mucins: GE, genetics
***Mucins: SE, secretion**
Oxidants: PD, pharmacology
RNA, Messenger: BI, biosynthesis
RNA, Messenger: GE, genetics
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Trachea: CY, cytology
Trachea: DE, drug effects
***Trachea: ME, metabolism**

CAS REGISTRY NO.: 68131-74-8 (fly ash); 7440-44-0 (Carbon)
CHEMICAL NAME: 0 (Antioxidants); 0 (Industrial Waste); 0 (Metals); 0 (Mucins); 0 (Oxidants); 0 (RNA, Messenger)

L136 ANSWER 2 OF 47 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 93076221 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1446252
TITLE: Tracheal epithelium in culture: a model for toxicity testing of inhaled molecules.
AUTHOR: Romet-Haddad S; Marano F; Blanquart C; Baeza-Squiban A
CORPORATE SOURCE: Laboratoire de Cytophysiologie et Toxicologie Cellulaire, Universite Paris, France.
SOURCE: Cell biology and toxicology, (1992 Jul-Sep) 8 (3) 141-50. Journal code: 8506639. ISSN: 0742-2091.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199212
ENTRY DATE: Entered STN: 19930129
Last Updated on STN: 19930129
Entered Medline: 19921229

ABSTRACT:
Rabbit trachea primary cultures have been developed as a model to evaluate the toxicity of noxious airborne pollutants. A mucociliary epithelium has been restored in vitro on collagen gel. Several general cytotoxicity assays (viability and growth inhibition) permit a first assessment for the acute toxicity of the tested molecules. More specific criteria such as measurement of the integrity of the epithelial barrier and inhibition of ciliary beat frequency allow to determine a specific impact of xenobiotics on the mucociliary epithelium in culture.

CONTROLLED TERM:
Acrolein: TO, toxicity
***Air Pollutants, Environmental: TO, toxicity**
Animals
Cell Division: DE, drug effects
Cell Survival: DE, drug effects
Cells, Cultured
Cilia: DE, drug effects
Electrodes
Epithelial Cells
Epithelium: DE, drug effects
Mechlorethamine: TO, toxicity
*Models, Biological
Rabbits
Research Support, Non-U.S. Gov't

L136 ANSWER 1 OF 47 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2000169401 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10702361
TITLE: Residual oil fly ash induces cytotoxicity and mucin secretion by guinea pig tracheal epithelial cells via an oxidant-mediated mechanism.
AUTHOR: Jiang N; Dreher K L; Dye J A; Li Y; Richards J H; Martin L D; Adler K B
CORPORATE SOURCE: Department of Anatomy, College of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina, USA.
CONTRACT NUMBER: HL 36982 (NHLBI)
SOURCE: Toxicology and applied pharmacology, (2000 Mar 15) 163 (3) 221-30.
Journal code: 0416575. ISSN: 0041-008X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000421
Last Updated on STN: 20021210
Entered Medline: 20000410

ABSTRACT:
Inhalation of ambient air particulate matter (PM) is associated with pulmonary injury and inflammation. Using primary cultures of guinea pig tracheal epithelial (GPTE) cells as an in vitro model of airway epithelium, we examined effects of exposure to suspensions of six different emission and ambient air PM samples: residual oil fly ash (ROFA) from an electrical power plant; fly ash from a domestic oil burning furnace (DOFA); ambient air dust from St. Louis (STL), Ottawa (OT), and Washington, DC (WDC); and volcanic ash from the eruption of Mount Saint Helens (MSH) in 1980. Effects of these particulates on cell viability (assessed via LDH assay), secretion of mucin (measured by a monoclonal antibody-based ELISA), and steady-state mRNA levels of the mucin gene MUC2 were determined. ROFA was the most toxic of the dusts tested, as it significantly increased LDH release following a 24-h incubation with 50 microg/cm(2) ROFA. ROFA also enhanced MUC2 mRNA after 4-h exposure, and mucin secretion after 8 h. ROFA-induced mucin secretion and cytotoxicity were attenuated by the oxidant scavenger, dimethylthiourea (DMTU). ROFA exposure also depleted cells of glutathione (GSH). Relatedly, depletion of intracellular GSH by treatment of the cells with buthionine sulfoxamine (BSO) also provoked mucin secretion, as well as enhancing the secretory effect of ROFA when the two agents were added together. L-NMA, the nitric oxide synthase (NOS) inhibitor, did not affect ROFA-induced mucin secretion. Of the soluble transition metals in ROFA (nickel, iron, vanadium), only vanadium individually, or combinations of the metals containing vanadium, provoked secretion. The results suggest ROFA enhances mucin secretion and generates toxicity in vitro to airway epithelium via a mechanism(s) involving generation of oxidant stress, perhaps related to depletion of cellular antioxidant capacity. Deleterious effects of inhalation of ROFA in the respiratory tract in vivo may relate to these cellular responses. Vanadium, a component of ROFA, may be important in generating these reactions.

CONTROLLED TERM: Animals
Antioxidants: PD, pharmacology
*Carbon: TO, toxicity
Cell Survival: DE, drug effects
Cells, Cultured
Dust: AE, adverse effects
Epithelial Cells: DE, drug effects

Searched by Barb O'Bryen, STIC 2-2518

Trachea: CY, cytology
***Trachea: DE, drug effects**

CAS REGISTRY NO.: 107-02-8 (Acrolein); 51-75-2 (Mechlorethamine)
CHEMICAL NAME: 0 (Air Pollutants, Environmental)

L136 ANSWER 3 OF 47 MEDLINE on STN
ACCESSION NUMBER: 2003057681 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12568494
TITLE: Mechanisms by which gram-positive bacteria and tobacco smoke stimulate mucin induction through the epidermal growth factor receptor (EGFR).
AUTHOR: Basbaum Carol; Li Daizong; Gensch Erin; Gallup Marianne; Lemjabbar Hassan
CORPORATE SOURCE: Biomedical Sciences Program, Department of Anatomy, University of California, San Francisco, CA 94143-0452, USA.
SOURCE: Novartis Foundation symposium, (2002) 248 171-6; discussion 176-80, 277-82.
Journal code: 9807767. ISSN: 1528-2511.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 20030206
Last Updated on STN: 20030529
Entered Medline: 20030528

ABSTRACT:
Mucin, the major macromolecular component of mucus, is generally considered to be a protective substance. When overproduced in a variety of lung diseases, however, mucin gives rise to clinical problems such as airway obstruction and recurrent infection. Our approach to identifying drug targets for the control of mucin overproduction is the analysis of cellular signalling pathways linking stimuli in the diseased lung to mucin transcription. Here we show that mucin transcription in response to both gram-positive bacteria and tobacco smoke is mediated through activation of the epidermal growth factor receptor (EGFR). The mode of activation of EGFR in response to bacterial lipoteichoic acid involves cleavage of the transmembrane ligand HBEGF by ADAM 10, whereas the activation of EGFR in response to smoke involves cleavage of amphiregulin by ADAM 17.

CONTROLLED TERM:
Cell Line
Endopeptidases: PH, physiology
Epithelial Cells: DE, drug effects
Epithelial Cells: ME, metabolism
Gene Expression Regulation: PH, physiology
Glycoproteins: ME, metabolism
*Gram-Positive Bacteria: PH, physiology
Humans
Intercellular Signaling Peptides and Proteins: ME, metabolism
Ligands
Lipopolysaccharides: PD, pharmacology
Lung: DE, drug effects
***Lung: ME, metabolism**
Metalloendopeptidases: PH, physiology
***Mucins: BI, biosynthesis**
Mucins: GE, genetics
Phosphorylation
Protein Processing, Post-Translational
Receptor, Epidermal Growth Factor: DE, drug effects

Searched by Barb O'Bryen, STIC 2-2518

*Receptor, Epidermal Growth Factor: PH, physiology
Receptors, Cell Surface
Signal Transduction
***Smoke**
Teichoic Acids: PD, pharmacology
Tobacco
Transcription, Genetic
src Homology Domains

CAS REGISTRY NO.: 117147-70-3 (amphiregulin); 56411-57-5 (lipoteichoic acid)
CHEMICAL NAME: 0 (Glycoproteins); 0 (Intercellular Signaling Peptides and Proteins); 0 (Ligands); 0 (Lipopolysaccharides); 0 (Mucins); 0 (Receptors, Cell Surface); 0 (Teichoic Acids); 0 (diphtheria toxin receptor); EC 2.7.1.112 (Receptor, Epidermal Growth Factor); EC 3.4.- (Endopeptidases); EC 3.4.23.- (secretase); EC 3.4.24 (Metalloendopeptidases); EC 3.4.24.- (TNF-alpha converting enzyme)

L136 ANSWER 4 OF 47 MEDLINE on STN
ACCESSION NUMBER: 2001246969 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11248147
TITLE: Rapid reduction of intracellular glutathione in human bronchial epithelial cells exposed to occupational levels of toluene diisocyanate.

AUTHOR: Lantz R C; Lemus R; Lange R W; Karol M H
CORPORATE SOURCE: Department of Cell Biology and Anatomy, The University of Arizona, Tucson, Arizona 85724, USA..
lantz@email.arizona.edu

CONTRACT NUMBER: 05651 (NIEHS)
ES06694
SOURCE: Toxicological sciences : an official journal of the Society of Toxicology, (2001 Apr) 60 (2) 348-55.
Journal code: 9805461. ISSN: 1096-6080.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010611
Last Updated on STN: 20010611
Entered Medline: 20010607

ABSTRACT:
Toluene diisocyanate (TDI) is a recognized chemical asthmogen, yet the mechanism of this toxicity and the molecular reactions involved have not been elucidated. We have previously shown that TDI vapor forms adducts with the apical surface of the respiratory epithelium, and that it colocalizes with ciliary tubulin. In vitro, we have shown rapid reaction of TDI with glutathione (GSH) and transfer of the bisGS-TDI adduct to a sulfhydryl-containing major histocompatibility complex peptide. This study sought to determine if intracellular GSH is altered following exposure to TDI. We used the dye CellTracker Green (chloromethylfluorescein, CMFDA) for detection of glutathione. One-day and 6-day air-liquid cultures of human bronchoepithelial cells (HBE) were exposed to 20-100 ppb TDI vapor for 5, 15, or 30 min. Cells were subsequently imaged using a confocal microscope. Both 1- and 6-day cultures showed a decrease in intensity of the thiol staining as a function of the TDI exposure dose. Doses as low as 20 ppb, the current permissible exposure limit (PEL) to TDI, resulted in rapid (within 5 min) decreases in fluorescence. The decreased fluorescence was not due to cytotoxicity or decrease in either esterase or glutathione-S-transferase activity, enzymes necessary for activation of the fluorescence of CMFDA. The decrease in glutathione levels was verified using another fluorescent label,

ThioGlo(TM) 1, and cell extracts. In addition, the mucus produced by 6-day air-liquid interface HBE cells in response to TDI exposure appeared to be protective, as HBE cells underlying mucus retained more fluorescence than did cells in the same cultures that were not covered with mucus. These results, along with previous data, strongly suggest that TDI enters pulmonary cells and reacts rapidly with intracellular GSH, and that this can occur at the current PEL of 20 ppb. This rapid reaction suggests the importance of cellular thiols in TDI-induced pulmonary disease.

CONTROLLED TERM: ***Air Pollutants, Occupational: TO, toxicity**
***Bronchi: DE, drug effects**
Bronchi: ME, metabolism
Bronchi: PA, pathology
Cells, Cultured
Dose-Response Relationship, Drug
***Epithelial Cells: DE, drug effects**
Epithelial Cells: ME, metabolism
Epithelial Cells: PA, pathology
Fluoresceins: ME, metabolism
Fluorescence
Fluorescent Dyes: ME, metabolism
Glutathione: DE, drug effects
*Glutathione: ME, metabolism
Humans
Microscopy, Confocal
Mucins: ME, metabolism
Research Support, U.S. Gov't, P.H.S.
Time Factors
*Toluene 2,4-Diisocyanate: TO, toxicity

CAS REGISTRY NO.: 136832-63-8 (5-chloromethylfluorescein); 584-84-9 (Toluene 2,4-Diisocyanate); 70-18-8 (Glutathione)

CHEMICAL NAME: 0 (Air Pollutants, Occupational); 0 (Fluoresceins); 0 (Fluorescent Dyes); 0 (Mucins)

L136 ANSWER 5 OF 47 MEDLINE on STN
ACCESSION NUMBER: 2000105580 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10637131
TITLE: Lung mucin production is stimulated by the air pollutant residual oil fly ash.
AUTHOR: Longphre M; Li D; Li J; Matovinovic E; Gallup M; Samet J M; Basbaum C B
CORPORATE SOURCE: Department of Anatomy and Cardiovascular Research Institute, University of California, San Francisco 94143, USA.
CONTRACT NUMBER: P01HL24136 (NHLBI)
R01HL43762 (NHLBI)
SOURCE: Toxicology and applied pharmacology, (2000 Jan 15) 162 (2) 86-92.
Journal code: 0416575. ISSN: 0041-008X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200003
ENTRY DATE: Entered STN: 20000314
Last Updated on STN: 20000314
Entered Medline: 20000302

ABSTRACT:
Human and animal exposure to particulate air pollution is correlated with airway mucus hypersecretion and increased susceptibility to infection. Seeking clues to the mechanisms underlying this pathology, we examined the effect of

the particulate air pollutant residual oil fly ash (ROFA) on production of the major component of mucus, mucin, and the major antibacterial protein of the respiratory tract, lysozyme. We found that following in vitro exposure to ROFA, epithelial cells showed an increase in mucin (MUC5AC) and lysozyme (LYS) steady state mRNA. This upregulation was controlled at least partly at the level of transcription as shown by reporter assays. Experiments testing the ability of the major components of ROFA to mimic these effects showed that vanadium, a metal making up 18.8% by weight, accounted for the bulk of the response. A screen of signaling inhibitors showed that MUC5AC and LYS induction by ROFA are mediated by dissimilar signaling pathways, both of which are, however, phosphotyrosine dependent. Recognizing that the ROFA constituent vanadium is a potent tyrosine phosphatase inhibitor and that mucin induction by pathogens is phophotyrosine dependent, we suggest that vanadium-containing air pollutants trigger disease-like conditions by unmasking phosphorylation-dependent pathogen resistance pathways.

CONTROLLED TERM:
***Air Pollutants, Environmental: TO, toxicity**
Bronchi: CY, cytology
Bronchi: DE, drug effects
Cell Line
Cytoplasm: DE, drug effects
Cytoplasm: ME, metabolism
Cytoplasm: PH, physiology
Dose-Response Relationship, Drug
Epithelial Cells: DE, drug effects
*Fuel Oils: TO, toxicity
Humans
***Lung: DE, drug effects**
***Lung: ME, metabolism**
Lung Neoplasms
***Mucins: BI, biosynthesis**
Particle Size
RNA, Messenger: ME, metabolism
Research Support, U.S. Gov't, P.H.S.
Signal Transduction: DE, drug effects
Transcription, Genetic: DE, drug effects
Tumor Cells, Cultured
*Vanadium: TO, toxicity

CAS REGISTRY NO.: 7440-62-2 (Vanadium)
CHEMICAL NAME: 0 (Air Pollutants, Environmental); 0 (Mucins); 0 (RNA, Messenger)

L136 ANSWER 6 OF 47 MEDLINE on STN
ACCESSION NUMBER: 1999036712 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9819294
TITLE: Comparison of ciliary activity and inflammatory mediator release from bronchial epithelial cells of nonatopic nonasthmatic subjects and atopic asthmatic patients and the effect of diesel exhaust particles in vitro.
AUTHOR: Bayram H; Devalia J L; Khair O A; Abdelaziz M M; Sapsford R J; Sagai M; Davies R J
CORPORATE SOURCE: Academic Department of Respiratory Medicine, St Bartholomew's and the Royal London School of Medicine and Dentistry, The London Chest Hospital, London E2 9JX, UK.
SOURCE: Journal of allergy and clinical immunology, (1998 Nov) 102 (5) 771-82.
Journal code: 1275002. ISSN: 0091-6749.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

Searched by Barb O'Bryen, STIC 2-2518

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981209

ABSTRACT:
BACKGROUND: Recent studies have suggested that asthmatic patients may be more susceptible to the adverse effects of air pollutants, including diesel exhaust particles (DEP). The underlying mechanisms, however, are not clear. METHODS: We cultured bronchial epithelial cells from bronchial biopsy specimens of well-characterized groups of nonatopic, nonasthmatic individuals and atopic patients with mild asthma and compared the ciliary beat frequency (CBF) and release of IL-8, GM-CSF, regulated on activation, normal T-cell expressed and secreted (RANTES), and soluble intercellular adhesion molecule-1 (sICAM-1) from these cells both before and after exposure for 24 hours to 10 to 100 micrograms/mL DEP in vitro. RESULTS: The baseline CBF was not found to be significantly different in the bronchial epithelial cell cultures of nonasthmatic and asthmatic individuals. Incubation with DEP significantly attenuated the CBF of both the nonasthmatic and asthmatic bronchial epithelial cell cultures at all concentrations of DEP investigated and were maximal (55.5% and 45.2%, respectively) after incubation with 100 micrograms/mL DEP. The bronchial epithelial cell cultures of asthmatic patients constitutively released significantly greater amounts of IL-8, GM-CSF, and sICAM-1 than bronchial epithelial cell cultures of nonasthmatic subjects. The cultures of only asthmatic patients additionally released RANTES. Incubation of the asthmatic cultures with 10 micrograms/mL DEP significantly increased the release of IL-8 (from 102.0 to 167.8 pg/micrograms cellular protein; P <.01), GM-CSF (from 0.43 to 1.87 pg/micrograms cellular protein; P <.01), and sICAM-1 (from 14.7 to 38.1 pg/micrograms cellular protein; P <.02) after 24 hours. Incubation with 50 to 100 micrograms/mL DEP, however, significantly decreased the release of IL-8 and RANTES from these cultures. In contrast, only the higher concentrations of 50 to 100 micrograms/mL DEP significantly increased release of IL-8 (from 37.9 to 71.5 pg/micrograms cellular protein; P <.05) and GM-CSF (from 0.06 to 0. 34 pg/micrograms cellular protein; P <.05) from the bronchial epithelial cells of nonasthmatic individuals. CONCLUSIONS: These results suggest that bronchial epithelial cells of asthmatic subjects are different from bronchial epithelial cells of nonasthmatic subjects with regard to the amounts and types of proinflammatory mediators they can release and that the increased sensitivity of bronchial epithelial cells of asthmatic subjects to DEP may possibly result in exacerbation of their disease symptoms.

CONTROLLED TERM: Check Tags: Comparative Study; Female; Male
Adult
*Asthma: ME, metabolism
*Asthma: PP, physiopathology
***Bronchi: CY, cytology**
Cells, Cultured
***Cilia: PH, physiology**
Epithelial Cells: CY, cytology
Humans
*Hypersensitivity, Immediate: ME, metabolism
*Hypersensitivity, Immediate: PP, physiopathology
*Inflammation Mediators: ME, metabolism
Lung Diseases, Obstructive: ME, metabolism
Lung Diseases, Obstructive: PP, physiopathology
Research Support, Non-U.S. Gov't
***Vehicle Emissions: AE, adverse effects**

CHEMICAL NAME: 0 (Inflammation Mediators); 0 (Vehicle Emissions)

L136 ANSWER 7 OF 47 MEDLINE on STN
ACCESSION NUMBER: 1998158681 MEDLINE

Searched by Barb O'Bryen, STIC 2-2518

DOCUMENT NUMBER: PubMed ID: 9490663
TITLE: The effect of diesel exhaust particles on cell function and release of inflammatory mediators from human bronchial epithelial cells in vitro.
AUTHOR: Bayram H; Devalia J L; Sapsford R J; Ohtoshi T; Miyabara Y; Sagai M; Davies R J
CORPORATE SOURCE: Academic Department of Respiratory Medicine, St. Bartholomew's and the Royal London School of Medicine and Dentistry, The London Chest Hospital, London, United Kingdom.
SOURCE: American journal of respiratory cell and molecular biology, (1998 Mar) 18 (3) 441-8.
Journal code: 8917225. ISSN: 1044-1549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980410
Last Updated on STN: 19980410
Entered Medline: 19980331

ABSTRACT:
Animal studies have reported that diesel exhaust particles (DEP), which constitute an important fraction of particulate air pollution, lead to inflammation and/or damage of the airways. To investigate the mechanisms underlying DEP-induced airway disease in humans, we have cultured human bronchial epithelial cells (HBEC) from surgically obtained bronchial explants and investigated the effects of purified DEP on the permeability and ciliary beat frequency (CBF) of HBEC, and on the release of inflammatory mediators from these cells. Exposure to 10-100 microg/ml DEP and a filtered solution of 50 microg/ml DEP significantly increased the electrical resistance of the cultures, reaching a maximum of 200% over baseline after 6 h incubation with 100 microg/ml DEP. In contrast, movement of 14C-labeled bovine serum albumin across cell cultures was not significantly altered by incubation of HBEC with DEP. Exposure to 50 microg/ml DEP, filtered DEP solution, and 100 migrog/ml DEP significantly attenuated the CBF of these cells by 51%, 33%, and 73%, respectively, from baseline after 24 h incubation. Similarly, 50 microg/ml DEP, filtered DEP solution, and 100 microg/ml DEP significantly increased the release of interleukin-8 from 12.9 pg/microg cellular protein to 41.6, 114.9, and 44.3 pg/microg cellular protein, respectively, after 24 h incubation. The release of granulocyte-macrophage colony stimulating factor (GM-CSF) and soluble intercellular adhesion molecule-1 (sICAM-1) was also significantly increased after exposure for 24 h to 50 microg/ml DEP (GM-CSF from 0.033 pg/microg cellular protein to 0.056 pg/mug cellular protein and sICAM-1 from 7.2 pg/microg cellular protein to 12.5 pg/microg cellular protein). These results suggest that exposure of HBEC to DEP may lead to adverse functional changes and release of proinflammatory mediators from these cells, and that these effects may influence the development of airway disease.

CONTROLLED TERM:
Adult
Air Pollutants, Environmental: CH, chemistry
***Air Pollutants, Environmental: TO, toxicity**
Bronchi: CY, cytology
***Bronchi: IM, immunology**
Cell Membrane Permeability: DE, drug effects
Cells, Cultured
Cilia: DE, drug effects
Epithelial Cells: CY, cytology
***Epithelial Cells: IM, immunology**
Granulocyte-Macrophage Colony-Stimulating Factor: SE, secretion

Searched by Barb O'Bryen, STIC 2-2518

Humans
*Inflammation Mediators: ME, metabolism
Intercellular Adhesion Molecule-1: BI, biosynthesis
Interleukin-8: SE, secretion
Lung Diseases: ET, etiology
Middle Aged
Polycyclic Compounds: TO, toxicity
Research Support, Non-U.S. Gov't
***Vehicle Emissions: TO, toxicity**

CAS REGISTRY NO.: 126547-89-5 (Intercellular Adhesion Molecule-1); 83869-56-1 (Granulocyte-Macrophage Colony-Stimulating Factor)

CHEMICAL NAME: 0 (Air Pollutants, Environmental); 0 (Inflammation Mediators); 0 (Interleukin-8); 0 (Polycyclic Compounds); 0 (Vehicle Emissions)

L136 ANSWER 8 OF 47 MEDLINE on STN
ACCESSION NUMBER: 93310724 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8322244
TITLE: Effect of azelastine on sulphur dioxide induced impairment of ciliary motility in airway epithelium.
AUTHOR: Tamaoki J; Chiyotani A; Sakai N; Takeyama K; Konno K
CORPORATE SOURCE: First Department of Medicine, Tokyo Women's Medical College, Japan.
SOURCE: Thorax, (1993 May) 48 (5) 542-6.
Journal code: 0417353. ISSN: 0040-6376.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199308
ENTRY DATE: Entered STN: 19930813
Last Updated on STN: 19930813
Entered Medline: 19930803

ABSTRACT:
OBJECTIVE--The effect of azelastine on airway mucociliary transport function was studied by measuring ciliary motility of human bronchial epithelium in vitro with a photoelectric method. METHOD--Bronchial epithelial cells were obtained by fibreoptic bronchoscopy, mounted in a Rose chamber, and perfused with Krebs-Henseleit solution. The preparations were placed on a microscope stage equipped with an illuminator, and the variations of light intensity caused by ciliary beating were detected by a photometer. RESULTS--The addition of azelastine to the perfusate increased ciliary beat frequency (CBF) in a dose dependent manner without ciliary discoordination. The mean (SE) maximal increase from the baseline value and the concentration required to produce a half maximal effect were 27.0 (4.2)% and 9.2 x 10(-6) mol/l, respectively. Exposure of the cells to the perfusate containing 3 ppm sulphur dioxide rapidly decreased CBF by 59.2 (5.0)%, and was accompanied by a reduction in intracellular cyclic AMP levels from 38.1 (4.3) to 10.1 (2.4) pmol/mg protein. This effect was prevented by pretreatment of cells with azelastine in a dose dependent manner. CONCLUSIONS--Azelastine not only stimulates ciliary motility of airway epithelium and hence mucociliary transport function, but may also protect against sulphur dioxide induced ciliary dysfunction, probably by inhibiting intracellular cyclic AMP loss.

CONTROLLED TERM: ***Bronchi: DE, drug effects**
*Bronchodilator Agents: PD, pharmacology
Cells, Cultured
***Cilia: DE, drug effects**
Epithelial Cells
Epithelium: DE, drug effects
Humans

Searched by Barb O'Bryen, STIC 2-2518

Mucociliary Clearance: DE, drug effects
Photometry
*Phthalazines: PD, pharmacology
Research Support, Non-U.S. Gov't
Stimulation, Chemical
Sulfur Dioxide: PD, pharmacology

CAS REGISTRY NO.: 58581-89-8 (azelastine); 7446-09-5 (Sulfur Dioxide)
CHEMICAL NAME: 0 (Bronchodilator Agents); 0 (Phthalazines)

L136 ANSWER 9 OF 47 MEDLINE on STN
ACCESSION NUMBER: 89007987 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3170450
TITLE: Ciliated cells in vitamin A-deprived cultured hamster tracheal epithelium do divide.
AUTHOR: Rutten A A; Beems R B; Wilmer J W; Feron V J
CORPORATE SOURCE: TNO-CIVO Toxicology and Nutrition Institute, Zeist, Netherlands.
SOURCE: In vitro cellular & developmental biology : journal of the Tissue Culture Association, (1988 Sep) 24 (9) 931-5. Journal code: 8506951. ISSN: 0883-8364.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198811
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881110

ABSTRACT:
The pseudostratified tracheal epithelium, composed of a heterogeneous phenotypically varying cell population, was studied with respect to the in vitro cell proliferative activity of differentiated epithelial cells. Ciliated tracheal epithelial cells so far have been considered to be terminally differentiated, nonproliferating cells. Tracheal organ cultures obtained from vitamin A-deprived Syrian Golden hamsters were cultured in a vitamin A-deficient, serum-free, hormone-supplemented medium. In vitamin A-deprived tracheal epithelium treated with physiologically active all-trans retinol and low cigarette-smoke condensate concentrations it is possible to stimulate the cell proliferation of both basal and columnar cells. Therefore, the probability of finding proliferating columnar cells was increased compared with the in vivo and the vitamin A-deprived situation in which cell proliferative activity is relatively low. In the presence of cigarette-smoke condensate in a noncytotoxic concentration, basal, small mucous granule, ciliated, and indifferent tracheal epithelial cells incorporated [methyl-3H]-thymidine into the DNA during the S phase. The finding that ciliated cells were labeled was supported by serial sections showing the same labeled ciliated cell in two section planes separated by 2 to 3 micron, without labeled epithelial cells next to the ciliated cell. Furthermore, a ciliated tracheal epithelial cell incorporating [methyl-3H]thymidine into DNA was also seen in tracheal cultures of vitamin A-deprived hamsters treated with all-trans retinol in a physiologic concentration.

CONTROLLED TERM:
Animals
Autoradiography
Cell Differentiation: DE, drug effects
Cell Division: DE, drug effects
Cells, Cultured
Cilia
Epithelial Cells
Epithelium: DE, drug effects
Hamsters

Searched by Barb O'Bryen, STIC 2-2518

Mesocricetus
Plants, Toxic
Research Support, Non-U.S. Gov't
Smoke
Tobacco
***Trachea: CY, cytology**
Trachea: DE, drug effects
Vitamin A: PD, pharmacology
*Vitamin A Deficiency: PA, pathology

CAS REGISTRY NO.: 11103-57-4 (Vitamin A)

L136 ANSWER 10 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:14199 CAPLUS
DOCUMENT NUMBER: 140:265914
TITLE: Responses of **cultured** human airway epithelial cells treated with diesel exhaust extracts will vary with the engine load
AUTHOR(S): Madden, Michael C.; Dailey, Lisa A.; Stonehuerner, Jacqueline G.; Harris, D. Bruce
CORPORATE SOURCE: Natl. Health and Environmental Effects Research Laboratory, ORD, U.S. Environmental Protection Agency, Research Triangle Park, NC, USA
SOURCE: Journal of Toxicology and Environmental Health, Part A (2003), 66(24), 2281-2297
CODEN: JTEHF8; ISSN: 1528-7394
PUBLISHER: Taylor & Francis, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 09 Jan 2004
AB Epidemiol. evidence suggests that increased morbidity and mortality are associated with the concns. of ambient air particulate matter (PM). Many sources contribute to the particulate fraction of ambient pollution, including diesel exhaust particulates (DEP). Diesel exhaust also contributes gas-phase pollutants to the atmospheric, and gaseous copollutants may influence the toxicity of PM. The composition of diesel exhaust varies greatly depending on the engine load conditions as well as other factors. To determine whether different diesel exhaust composition can affect lung cell responses, the effects of diesel exhaust exts. derived from different engine loads were examined on normal human bronchial epithelial cells (NHBE) in vitro. Diesel exhaust was collected into chilled impingers containing phosphate-buffered saline (PBS). Cultured NHBE cells were treated with 0 to 500 μg/well of extract from .apprx.0% engine load (termed low load of LL) or extract from .apprx.75% engine load (termed high load or HL) for 24h. The HL extract was cytotoxic at 500 μg compared to controls as measured by 51Cr release. Production of the neutrophil chemotaxin interleukin 8 (IL-8) was decreased 4.7-fold in cells treated with 500 μgLL extract, whereas cells treated with 500 μg HL extract showed a 2.4-fold increase in IL-8 release. Production of the inflammatory and immune system mediator prostaglandin E2 (PGE2) was increased up to 2.5-fold in cells treated with HL extract, but unchanged with other treatments. Melittin stimulation of cells showed that the LL extract had an inhibitory effect on PGE2 release at 500 μg. Differences in carbonyl content of the exts. were found by high-performance liquid chromatog./mass spectrometry (HPLC/MS), with the HL extract having more intermediate size carbonyls (i.e., with six to nine carbons). The data suggest that the response of NHBE cells to treatment with diesel exhaust will vary depending on the constituent components of

the exhaust.
CC 4-3 (Toxicology)
Section cross-reference(s): 59
IT Aldehydes, biological studies
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(C2-C12; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
IT Exhaust gases (engine)
(diesel, extract; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
IT **Respiratory** system
(**epithelium**; responses of **cultured** human airway **epithelial** cells treated with diesel exhaust exts. will vary with engine load)
IT **Air pollution**
(exhaust; responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
IT **Epithelium**
(**respiratory** tract; responses of **cultured** human airway **epithelial** cells treated with diesel exhaust exts. will vary with engine load)
IT Human
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
IT Interleukin 8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
IT 363-24-6, Prostaglandin E2 329900-75-6, Cyclooxygenase-2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(responses of **cultured** human airway epithelial cells treated with diesel exhaust exts. will vary with engine load)
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 11 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 1999:302795 CAPLUS
DOCUMENT NUMBER: 131:69365
TITLE: Evaluation of two in vitro **ciliated** **epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation
AUTHOR(S): Swann, J. M.; Kennedy, J. R.; Schultz, T. W.
CORPORATE SOURCE: Department of Biochemistry, Cellular, and Molecular Biology, The University of Tennessee, Knoxville, TN, 37996-4500, USA
SOURCE: In Vitro & Molecular Toxicology (1999), 12(1), 17-32
CODEN: IVMTFJ; ISSN: 1097-9336
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 18 May 1999
AB Two aquatic-based in vitro ciliated epithelial systems, dog trachea and frog palate, have been evaluated as potential screens for sensory irritation of inhaled chems. Initially, the effects of a series of nonpolar narcotic agents (i.e., alcs. and ketones) were assessed by monitoring **ciliary beat** frequency under standard (static, 5-min exposure) conditions with the concentration (M) eliciting a 50% reduction in

ciliary beat frequency (i.e., EC50) being determined These data along with 1-octanol per water partition coefficient (log KOW) were used to formulate baseline predictive models. The minimal toxicity model for the canine trachea (ct) system is log 1/ctEC50 = 0.79 (log KOW) - 0.002; n = 7, r2 = 0.987, s = 0.15, F = 373, Pr > F = 0.0001. The frog palate (fp) baseline toxicity equation is log 1/fpEC50 = 0.80 (log KOW) - 0.12; n = 7, r2 = 0.995, s = 0.09, F = 1112, Pr > F = 0.0001. Prevalidation of both in vitro systems were undertaken by regression analyses with in vivo mouse inhalation toxicity data. The slopes of both analyses are not statistically different than 1.0. The intercepts reveal the in vivo endpoint to be tenfold more sensitive. Also, a set of bioreactive chems. was tested. Mechanisms of toxicity represented by this set of chems. included three specific electrophilic or nucleophilic mechanisms, weak acid respiratory uncoupling, and reversible and irreversible acetylcholinesterase inhibition. Although the relative ranking varied between the two in vitro systems, all bioreactive chems. were to have toxicity greater than twice that predicted by the baseline models. Based on these preliminary data, we feel that such a **ciliary beat** frequency in vitro system would provide valuable hazard information about potential sensory irritation of inhaled chems.

CC **4-1** (Toxicology)

IT Chemicals
Palate
Simulation and Modeling, biological
Toxicity
Trachea (anatomical)
(evaluation of two in vitro **ciliated epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT Alcohols, biological studies
Ketones, biological studies
RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL (Biological study)
(evaluation of two in vitro **ciliated epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT **Toxicity**
(inhalation; evaluation of two in vitro **ciliated epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation)

IT 63-25-2, Carbaryl 64-17-5, Ethanol, biological studies 71-41-0, 1-Pentanol, biological studies 78-93-3, 2-Butanone, biological studies 100-44-7, Benzyl chloride, biological studies 107-87-9, 2-Pentanone 111-70-6, 1-Heptanol 111-71-7, Heptanal 111-87-5, 1-Octanol, biological studies 112-12-9, 2-Undecanone 121-75-5, Malathion 534-52-1, 4,6-Dinitro-o-cresol 693-54-9, 2-Decanone 2548-87-0
RL: ADV (Adverse effect, including toxicity); PRP (Properties); BIOL (Biological study)
(evaluation of two in vitro **ciliated epithelial** systems, dog trachea and frog palate, for potential as screens for sensory irritation)

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 12 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1996:88536 CAPLUS
DOCUMENT NUMBER: 124:138291
TITLE: The effects of the organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro

AUTHOR(S): Swann, J. M.; Schultz, T. W.; Kennedy, J. R.
CORPORATE SOURCE: Univ. Tennessee, Coll. Veterinary Med., Knoxville, TN, 37996, USA
SOURCE: Archives of Environmental Contamination and Toxicology (1996), 30(2), 188-94
CODEN: AECTCV; ISSN: 0090-4341
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 10 Feb 1996
AB The ciliotoxic potential of the organophosphorous insecticides Dursban and Lorsban, their active ingredient, chlorpyrifos, and their carrier ingredients (Blanks) were assessed. Since chlorpyrifos inhibits acetylcholinesterase, the acetylcholine-innervated ciliated epithelial cultures of frog palate were used as the model. All compds. caused a decrease in frequency of **ciliary beat** over time. EC50 values followed the same order as the time to inhibition. The orders were Lorsban > Dursban > chlorpyrifos, and Lorsban > Dursban .apprx. Lorsban Blank > Dursban Blank. Stimulation of **ciliary beating** occurred immediately after exposure to all compds., followed by inhibition. Dursban, Lorsban, and both Blanks elicited stimulatory effects in the presence of atropine. Atropine only blocked the initial stimulatory response with chlorpyrifos. In addition to chlorpyrifos, some component(s) of the inert ingredients were initially stimulatory but ultimately inhibitory to **ciliary beating** in the frog palate model. All compds. caused mitochondrial damage, including swelling, disruption of cristae, and loss of matrix.
CC **4-4** (Toxicology)
ST organophosphorous insecticide **ciliated epithelium** palate frog; dursban lorsban chlorpyrifos **ciliated epithelium** frog; Rana organophosphorous insecticide **ciliated epithelium**
IT Cell morphology
Insecticides
Mitochondria
Rana pipiens
Toxicity
(toxicity of organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro)
IT **Epithelium**
(**ciliated**, toxicity of organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro)
IT Palate
(**epithelium**, toxicity of organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro)
IT Organic compounds, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(phosphorus-containing, toxicity of organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro)
IT 2921-88-2, Dursban
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of organophosphorus insecticides Dursban and Lorsban on the **ciliated epithelium** of the frog plate in vitro)
IT 51-55-8, Atropine, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(toxicity of organophosphorus insecticides Dursban and Lorsban on the

ciliated epithelium of the frog plate in vitro)

L136 ANSWER 13 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:968536 CAPLUS
DOCUMENT NUMBER: 143:481862
TITLE: Influence of seasons and sampling strategy on assessment of bioaerosols in sewage treatment plants in Switzerland
AUTHOR(S): Oppliger, Anne; Hilfiker, Silvia; Duc, Trinh Vu
CORPORATE SOURCE: Institute of Occupational Health Sciences, Lausanne, CH-1015, Switz.
SOURCE: Annals of Occupational Hygiene (2005), 49(5), 393-400
CODEN: AOHYA3; ISSN: 0003-4878
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 06 Sep 2005
AB An assessment of sewage workers' exposure to airborne cultivable bacteria, fungi and inhaled endotoxins was performed at 11 sewage treatment plants. We sampled the enclosed and unenclosed treatment areas in each plant and evaluated the influence of seasons (summer and winter) on bioaerosol levels. We also measured personal exposure to endotoxins of workers during special operation where a higher risk of bioaerosol inhalation was assumed. Results show that only fungi are present in significantly higher concns. in summer than in winter (2331 ± 858 vs. 329 ± 95 CFU m-3). We also found that there are significantly more bacteria in the enclosed area, near the particle grids for incoming water, than in the unenclosed area near the aeration basins (9455 ± 2661 vs. 2435 ± 985 CFU m-3 in summer and 11 081 ± 2299 vs. 2002 ± 839 CFU m-3 in winter). All bioaerosols were frequently above the recommended values of occupational exposure. Workers carrying out special tasks such as cleaning tanks were exposed to very high levels of endotoxins (up to 500 EU m-3) compared to routine work. The species composition and concentration of airborne Gram-neg. bacteria were also studied. A broad spectrum of different species within the Pseudomonadaceae and the Enterobacteriaceae families were predominant in nearly all plants investigated.
CC **59-5** (Air Pollution and Industrial Hygiene)
Section cross-reference(s): **4**, 10
IT Acinetobacter baumannii
Acinetobacter junii
Aeromonas hydrophila
Air pollution
Burkholderia cepacia
Chromobacterium violaceum
Chryseomonas luteola
Comamonas acidovorans
Enterobacter aerogenes
Enterobacter cloacae
Enterobacteriaceae
Escherichia coli
Fungi
Human
Industrial hygiene
Klebsiella oxytoca
Klebsiella pneumoniae
Occupational health hazard
Ochrobactrum anthropi
Pantoea agglomerans
Pseudomonadaceae
Pseudomonas aeruginosa

Pseudomonas indologenes
Pseudomonas oryzihabitans
Pseudomonas pseudoalcaligenes
Pseudomonas putida
Ralstonia pickettii
Raoultella terrigena
Salmonella choleraesuis arizonae
Sampling
Serratia ficaria
Serratia proteamaculans proteamaculans
Temperature
Wastewater treatment
(bioaerosols occupational exposure assessment in different season, for different work sites and during special tasks in wastewater treatment plant in Switzerland)

IT Eubacteria
(**culturable**; bioaerosols occupational exposure assessment in different season, for different work sites and during special tasks in wastewater treatment plant in Switzerland)

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 14 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:505432 CAPLUS
TITLE: Integrating measurements of radon and thoron and their deposition fractions in the respiratory tract
AUTHOR(S): Zhuo, W.; Tokonami, S.; Yonehara, H.; Yamada, Y.
CORPORATE SOURCE: Radon Research Group, National Institute of Radiological Sciences, Inage, Chiba, 263-8555, Japan
SOURCE: Radioactivity in the Environment (2005), 7(Natural Radiation Environment VII), 352-360
CODEN: REANCK
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 14 Jun 2005
AB For simultaneous measurements of indoor 222Rn and 220Rn and estimation of their deposition fractions in the respiratory tract, a new type of passive integrating 222Rn and 220Rn monitor and a portable bronchial dosimeter were developed. The passive 222Rn and 220Rn monitor was rebuilt from a com. available passive 222Rn monitor. Besides its simple construction, the volume and weight of the new monitor are only 110 cm3 and 20 g. Calibration factors of 222Rn and 220Rn for the new monitor were systematically studied through calibration expts. The results indicated that indoor 220Rn could be discriminated from 222Rn by using the new passive monitor. The bronchial dosimeter consists of three sets of progeny integrating sampling units (PISUs) with different configurations of sampling heads. Multiple metal screens are used to mimic the penetrating and deposition behavior of 222Rn/220Rn progeny in the nasal and tracheo-bronchial (T-B) regions of the human respiratory tract. The potential alpha energy concns. (PAEC) of 222Rn/220Rn progeny are directly measured with the allyl diglycol carbonate (CR-39) detectors inside the PISUs. The deposition fractions of 222Rn and 220Rn progeny in the T-B region were measured, with avs. of 4.5 and 4.0% for ordinary room conditions, in general agreement with other reported values. Both the new monitor and device are simple and compact as well as of low cost, and they are considered to be practical for large-scale and long-term surveys of indoor 222Rn and 220Rn.
CC 9 (Biochemical Methods)
IT INDEXING IN PROGRESS

IT **Respiratory** system
(**epithelium**; integrated measurements of radon and thoron and their deposition fractions in the **respiratory** tract)

IT **Biosensors**
(integrated measurements of radon and thoron and their deposition fractions in the respiratory tract using a bronchial dosimeter)

IT **Epithelium**
(**respiratory** tract; integrated measurements of radon and thoron and their deposition fractions in the **respiratory** tract)

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 15 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2005:337517 CAPLUS
DOCUMENT NUMBER: 143:119370
TITLE: Pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**
AUTHOR(S): Dagher, Zeina; Garcon, Guillaume; Gosset, Pierre; Ledoux, Frederic; Surpateanu, Georgiana; Courcot, Dominique; Aboukais, Antoine; Puskaric, Emile; Shirali, Pirouz
CORPORATE SOURCE: Laboratoire de Recherche en Toxicologie Industrielle et Environnementale, Maison de la Recherche en Environnement Industriel de Dunkerque 2, Dunkerque, 59140, Fr.
SOURCE: Journal of Applied Toxicology (2005), 25(2), 166-175
CODEN: JJATDK; ISSN: 0260-437X
PUBLISHER: John Wiley & Sons Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 20 Apr 2005
AB Exposure to urban airborne particulate matter (PM) has been associated with adverse health effects. The majority of research articles published on air pollution PM relate to PM10. However, increasing emphasis and stringent regulations have been placed on PM2.5. The mechanisms for PM-induced adverse health effects are not well understood, but inflammation seems to be of importance. We focused our attention also on the capacity of air pollution PM2.5 to induce cytotoxic and inflammatory responses in human epithelial lung cells (L132) in culture. Particulate matter was collected in Dunkerque, a French seaside city characterized by the proximity of industrial activity and heavy motor vehicle traffic. Size distribution results showed that the cumulative frequency of PM2.5 was 92.15% and their sp. surface area was 1 m2 g-1. Inorg. and organic chems. usually associated with the natural environment but also so-called anthropogenic elements were found in PM, suggesting that much of the PM was derived from wind-borne dust from the industrial complex and the heavy diesel motor vehicle. We observed PM concentration-dependent cytotoxic effects in L132 cells (LC10 = 18.84 μg PM ml-1; LC50 = 75.36 μg PM ml-1). We showed that exposure to Dunkerque City's PM2.5 induced significant increases (in a concentration- and time-dependent manner) in protein secretion and/or gene expression of inflammatory cytokines (i.e. TNF-α, IL-1β, IL-8, GM-CSF, IL-6, TGF-β1). We hypothesized also that the occurrence of the acute inflammatory response might rely on the capacity of such air pollutants to generate oxidative species, which have been implicated in the stringent regulation of the cytokine network. Hence, we suggest that the development of inflammatory effects that worsen over time stems from the cytotoxicity in Dunkerque City's PM2.5-exposed

L132 cells in culture.
CC 59-2 (Air Pollution and Industrial Hygiene)
Section cross-reference(s): 4
IT Lung
(epithelium; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Waste gases
(industrial; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT **Respiratory** system, disease
(inflammation; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human **epithelial** lung cells (L132) in **culture**)
IT **Air pollution**
(particulate; pro-inflammatory effects of dunkerque city **air** pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Cytotoxicity
Exhaust gases (engine)
Human
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Interleukin 1β
Interleukin 6
Interleukin 8
Tumor necrosis factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Health hazard
(pro-inflammatory effects of dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Epithelium
(pulmonary; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Inflammation
(**respiratory** tract; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human **epithelial** lung cells (L132) in **culture**)
IT Airborne particles
(urban; pro-inflammatory effects of dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT Transforming growth factors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(β1-; pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
IT 83869-56-1, GM-CSF,
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(pro-inflammatory effects of Dunkerque city air pollution particulate matter 2.5 in human epithelial lung cells (L132) in **culture**)
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 16 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:100900 CAPLUS

DOCUMENT NUMBER: 140:132674
TITLE: Real-time detection method and system for identifying individual aerosol particles
INVENTOR(S): Gard, Eric Evan; Fergenson, David Philip
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: U.S. Pat. Appl. Publ., 16 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004024539	A1	20040205	US 2002-280608	20021024
US 6959248	B2	20051025		
US 2005073683	A1	20050407	US 2004-916737	20040811
			US 2001-335598P P	20011025
			US 2002-280608 A2	20021024
			US 2003-494442P P	20030811

PRIORITY APPLN. INFO.:

ED Entered STN: 08 Feb 2004
AB A method and system of identifying individual aerosol particles in real time. Sample aerosol particles are compared against and identified with substantially matching known particle types by producing pos. and neg. test spectra of an individual aerosol particle using a bipolar single particle mass spectrometer. Each test spectrum is compared to spectra of the same resp. polarity in a database of predetd. pos. and neg. spectra for known particle types and a set of substantially matching spectra is obtained. Finally the identity of the individual aerosol particle is determined from the set of substantially matching spectra by determining a best matching one of the known particle types having both a substantially matching pos. spectrum and a substantially matching neg. spectrum associated with the best matching known particle type.
IC ICM G06F019-00
ICS G01N031-00
INCL 702028000
CC **59-2** (Air Pollution and Industrial Hygiene)
Section cross-reference(s): **4**, 10
IT **Airborne particles**
(aerosols, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)
IT **Culture** media
(inclusion in sample; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)
IT **Air pollution**
(particulate, aerosol, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)
IT Alarm devices
Biological warfare agents
Chemical warfare agents
Sampling
Time-of-flight mass spectrometry
(real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

L136 ANSWER 17 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:913421 CAPLUS

DOCUMENT NUMBER: 139:368744
TITLE: Real-time detection method and system for identifying individual aerosol particles

INVENTOR(S): Gard, Eric E.; Fergenson, David P.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003096375	A1	20031120	WO 2002-US34291	20021025

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-280608 A 20011025

ED Entered STN: 21 Nov 2003

AB A method and system of identifying individual aerosol particles in real time. Sample aerosol particles are compared against and identified with substantially matching known particle types by producing pos. and neg. test spectra of an individual aerosol particle using a bipolar single particle mass spectrometer. Each test spectrum is compared to spectra of the same resp. polarity in a database of predetd. pos. and neg. spectra for known particle types and a set of substantially matching spectra is obtained. Finally the identity of the individual aerosol particle is determined from the set of substantially matching spectra by determining a best matching one of the known particle types having both a substantially matching pos. spectrum and a substantially matching neg. spectrum associated with the best matching known particle type.

IC ICM H01J049-40
ICS H01J049-04; H01J049-16

CC **59-2** (Air Pollution and Industrial Hygiene)
Section cross-reference(s): 4, 10

IT **Airborne particles**
(aerosols, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT **Culture** media
(inclusion in sample; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT **Air pollution**
(particulate, aerosol, anal. of; real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)

IT Alarm devices
Biological warfare agents
Chemical warfare agents
Sampling
Time-of-flight mass spectrometry

(real-time detection method and system for identifying individual aerosol particles using bipolar single particle mass spectrometry)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 18 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:429352 CAPLUS
DOCUMENT NUMBER: 135:57096
TITLE: Effects of formaldehyde on the frog's mucociliary **epithelium** as a surrogate to evaluate air pollution effects on the **respiratory epithelium**
AUTHOR(S): Flo-Neyret, C.; Lorenzi-Filho, G.; Macchione, M.; Garcia, M. L. B.; Saldiva, P. H. N.
CORPORATE SOURCE: Departamento de Patologia Faculdade de Medicina, Universidade Federal de Sao Paulo, Sao Paulo, 01246-903, Brazil
SOURCE: Brazilian Journal of Medical and Biological Research (2001), 34(5), 639-643
CODEN: BJMRDK; ISSN: 0100-879X
PUBLISHER: Associacao Brasileira de Divulgacao Cientifica
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 14 Jun 2001
AB The increasing use of alc. as an alternative fuel to gasoline or diesel can increase emission of formaldehyde, an organic gas that is irritant to the mucous membranes. The respiratory system is the major target of air pollutants and its major defense mechanism depends on the continuous activity of the cilia and the resulting constant transportation of mucous secretion. The present study was designed to evaluate the effects of formaldehyde on the ciliated epithelium through a relative large dose range around the threshold limit value adopted by the Brazilian legislation, namely 1.6 ppm (1.25-5 ppm). For this purpose, the isolated frog palate preparation was used as the target of toxic injury. Four groups of frog palates were exposed to diluted Ringer solution (control, N = 8) and formaldehyde diluted in Ringer solution at 3 different concns. (1.25, 2.5, and 5.0 ppm, N = 10 for each group). Mucociliary clearance and **ciliary beat** frequency decreased significantly in contact with formaldehyde at the concns. of 2.5 and 5.0 ppm after 60 min of exposure ($P<0.05$). Thus, relatively low concns. of formaldehyde, which is even below the Brazilian threshold limit value, are sufficient to cause short-term mucociliary impairment.
CC **4-3** (Toxicology)
Section cross-reference(s): **59**
ST formaldehyde Rana palate cilia air pollution **respiratory epithelium**
IT **Air pollution**
Cilia
Palate
Rana catesbeiana
(effects of formaldehyde on frog mucociliary **epithelium** as surrogate to evaluate **air** pollution effects on **respiratory epithelium**)
IT **Respiratory** tract
(**epithelium**; effects of formaldehyde on frog mucociliary **epithelium** as surrogate to evaluate air pollution effects on **respiratory epithelium**)
IT 50-00-0, Formaldehyde, biological studies
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)

(effects of formaldehyde on frog mucociliary **epithelium** as surrogate to evaluate air pollution effects on **respiratory epithelium**)

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 19 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2001:431342 CAPLUS
DOCUMENT NUMBER: 135:176512
TITLE: The further development of rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicology risk assessment
AUTHOR(S): Dowling, K.; Mothersill, C.
CORPORATE SOURCE: Radiation Science Centre, Dublin Institute of Technology, Dublin, Ire.
SOURCE: Aquatic Toxicology (2001), 53(3-4), 279-289
CODEN: AQTODG; ISSN: 0166-445X
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 14 Jun 2001
AB The use of short-term cytotoxicity assays for the initial screening of chems. not only aids in establishing priorities for the selection of chems. that should be tested in vivo, but also decreases the time in which potential toxicants can be valued. Rainbow trout primary skin epithelial cell cultures are one such assay. Rainbow trout primary skin cell cultures contain two cell types, keratinocytes and goblet mucus cells. Two aquatic pollutants, copper and prochloraz were screened using this cell system. The influence of media composition on the effects of the aquatic pollutants was also studied by testing the chems. in both serum-containing and serum-free medium and the morphol. changes that occurred within the cell cultures recorded. The concentration of copper that causes a reduction of 90% in the residual of day 3 growth of the primary cell culture system was found to be approx. 10 fold more than that of prochloraz. Prochloraz was found to cause a greater reduction in growth area when added to the primary cell culture system in serum-free medium than in serum-containing medium. Copper, in contrast, was found to exert reduced toxicity when added to the test cultures in serum-free medium compared with addition in serum-containing medium. Prochloraz was found to kill the epithelial cells by a process of necrosis. Copper, was found to kill the epithelial cells by both necrosis and apoptosis in a ratio of 2:1. It was also observed that as the dose of both chems. increased, the number of goblet cells contained in the cell cultures decreased. A PAS stain was carried out to determine if the goblet cells were exocytosing their contents onto the cell culture surface. It was found that as chemical exposure increased the number of cells expressing positivity for mucus also increased. The results of this study add further evidence to support that primary cell cultures are a very appropriate model for toxicity risk assessment.
CC 4-1 (Toxicology)
Section cross-reference(s): 61
IT **Toxicity**
(aquatic; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)
IT Proteins, general, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(blood; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

IT Animal tissue **culture**
Apoptosis
Bioassay
Cell death
Cell morphology
Cytotoxicity
Diagnosis
Ecotoxicity
Environmental pollution
Exocytosis
Oncorhynchus mykiss
Risk assessment
Water pollution
Xenobiotics
(ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

IT Skin
(epithelium; ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

IT Skin
(**goblet** cell; ecotoxicity risk assessment in relation to rainbow trout primary **epithelial** cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

IT 7447-39-4, Cupric chloride, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

IT 7440-50-8, Copper, biological studies 67747-09-5, Prochloraz
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(ecotoxicity risk assessment in relation to rainbow trout primary epithelial cell **cultures** as a diagnostic tool in ecotoxicol. risk assessment)

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 20 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:8546 CAPLUS
DOCUMENT NUMBER: 132:218213
TITLE: Internalization of Carcinogenic Lead Chromate Particles by **Cultured** Normal Human Lung Epithelial Cells: Formation of Intracellular Lead-Inclusion Bodies and Induction of Apoptosis
AUTHOR(S): Singh, Jatinder; Pritchard, Daryl E.; Carlisle, Diane L.; Mclean, John A.; Montaser, Akbar; Orenstein, Jan M.; Patierno, Steven R.
CORPORATE SOURCE: Department of Pharmacology, George Washington University Medical Center, Washington, DC, 20037, USA
SOURCE: Toxicology and Applied Pharmacology (1999), 161(3), 240-248
CODEN: TXAPA9; ISSN: 0041-008X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

Searched by Barb O'Bryen, STIC 2-2518

ED Entered STN: 05 Jan 2000
AB Occupational exposure to certain particulate hexavalent chromium [Cr(VI)] compds., such as lead chromate, has been associated with lung cancer and respiratory tract toxicity. The authors have previously shown that apoptosis is a major mode of death in cultured rodent cells treated with soluble sodium chromate and particulate lead chromate. Here the authors report the cellular and mol. effects of lead chromate and sodium chromate in normal human lung small airway epithelial (HSAE) cells, which may be one of the targets for Cr(VI)-induced lung cancer and respiratory tract toxicity. Phagocytosed lead chromate particles and intracellular lead-inclusion bodies (LIB) were observed by transmission electron microscopy and confirmed by X-ray anal. HSAE cells exposed to lead chromate and sodium chromate underwent dose-dependent apoptosis. The cellular uptake and genomic interactions of both Cr and lead (Pb) were examined by inductively coupled plasma mass spectrometry (ICPMS) coupled with a novel, direct-injection high-efficiency nebulizer (DIHEN). Using this approach, the authors have quantitated a dose-dependent formation of Cr-DNA adducts and DNA-associated Pb in lead chromate-treated HSAE cells. The formation of LIB in normal human lung cells exposed to lead chromate indicates that ionic Pb is released from the particles and thus might contribute to the cell toxicity caused by lead chromate. Internalization and dissoln. of lead chromate particles and the interaction of ionic Cr and Pb with DNA, may be components of the mechanism of lead chromate carcinogenesis. Lead chromate-induced apoptosis may be a mechanism to eliminate cells with chromium- and/or lead-damaged DNA. (c) 1999 Academic Press.
CC 4-6 (Toxicology)
Section cross-reference(s): 59
IT DNA
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(adducts, with chromium; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(damage; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT Carcinogens
(environmental; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT Lung
(epithelium, cells; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT **Respiratory** tract
(**epithelium**; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT Mass spectrometry
(inductively coupled plasma; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)
IT **Air pollution**
(industrial; lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular

lead-inclusion bodies and induction of apoptosis)

IT Airborne particles
Apoptosis
Cell morphology
Cell nucleus
Cytoplasm
Cytotoxicity
Genotoxicity
Inclusion bodies
Lung, neoplasm
Occupational health
Transmission electron microscopy
X-ray spectroscopy
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT Toxicity
(pulmonary, **respiratory**; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT Lung
(toxicity, **respiratory**; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT **Respiratory** tract
(toxicity; lead chromate particle internalization by **cultured** normal human lung **epithelial** cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7775-11-3, Sodium chromate
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7440-47-3D, Chromium, adducts with DNA, biological studies
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

IT 7439-92-1, Lead, biological studies 7440-47-3, Chromium, biological studies 7758-97-6, Lead chromate 11104-59-9, Chromate 18540-29-9, biological studies
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(lead chromate particle internalization by **cultured** normal human lung epithelial cells and formation of intracellular lead-inclusion bodies and induction of apoptosis)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 21 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1997:315762 CAPLUS
DOCUMENT NUMBER: 126:326672
TITLE: The effect of bacterial toxins on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency
AUTHOR(S): Kanthakumar, K.; Taylor, G. W.; Cundell, D. R.;

CORPORATE SOURCE: Dowling, R. B.; Johnson, M.; Cole, P. J.; Wilson, R.
Host Defence Unit, Imperial Coll. Sci., Tech. and Med., National Heart and Lung Inst., London, SW3 6LR, UK

SOURCE: Pulmonary Pharmacology (1996), 9(4), 223-230
CODEN: PUPHEX; ISSN: 0952-0600
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 17 May 1997

AB The authors investigated whether reduction in intracellular adenosine nucleotides might be a common mechanism of action of other bacterial toxins which slow **ciliary beat.** Two other Pseudomonas aeruginosa toxins, 1-hydroxyphenazine (1-HP) and rhamnolipid, and two Haemophilus influenzae fractions produced by gel filtration of broth cultures were tested. The effect on human nasal epithelium **ciliary beat** frequency (CBF), and intracellular cAMP and ATP were measured, and the effect of the pharmacol. agents, dibutyryl cAMP and salmeterol, on these changes was assessed. 1-HP, rhamnolipid and the two H. influenzae fractions slowed CBF before there was significant release of lactate dehydrogenase from the cells. The toxins also caused a fall in intracellular cAMP and ATP. Dibutyryl cAMP and salmeterol at the concns. used do not increase baseline CBF, but diminished the fall in CBF and intracellular adenosine nucleotides. The cAMP and ATP levels in these studies were combined with those previously obtained with pyocyanin. There was a good correlation between cAMP and ATP levels and CBF. Bacterial toxins which slow CBF may act by causing a fall in intracellular adenosine nucleotides, and agents which stimulate cAMP may prevent toxin-induced slowing of **ciliary beat.**

CC **4-5** (Toxicology)
ST bacterial toxin adenosine nucleotide **ciliary beat**; nasal **ciliated epithelium** human bacterial toxin
IT Haemophilus influenzae
Toxicity
(bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT Toxins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bacterial; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT Nose
(**epithelium, ciliated**; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT Cilia
(nasal epithelium; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT Glycolipids
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(rhamnose-containing; bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT 528-71-2, 1-Hydroxyphenazine
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
IT 56-65-5, 5'-ATP, biological studies 58-61-7D, Adenosine, nucleotides, biological studies 60-92-4, CAMP

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bacterial toxins effects on levels of intracellular adenosine nucleotides and human **ciliary beat** frequency)
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L136 ANSWER 22 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:762561 CAPLUS
DOCUMENT NUMBER: 126:43660
TITLE: In vitro reconstituted tissue as an alternative to human respiratory tract
AUTHOR(S): Emura, M.; Ochiai, A.; Hirohashi, S.
CORPORATE SOURCE: Inst. Experimentelle Pathologie, Med. Hochschule, Hannover, D-30625, Germany
SOURCE: Toxicology Letters (1996), 88(1-3), 81-84
CODEN: TOLED5; ISSN: 0378-4274
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 01 Jan 1997
AB The data obtained from in vitro systems utilizing human cells and tissues should form a basic part of the information necessary for risk assessment. The most important thing for such systems is, therefore, to simulate the structures and functions of cells and tissues in the native organ as closely as possible. In designing in vitro systems, there may be two approaches-one aiming at the growth of cells in a primarily two-dimensional fashion, the other allowing cells to form in vivo-mimicking three-dimensional architectures. In cultures in which the airway epithelial cells are growing in a two-dimensional fashion, some functional and structural characteristics can be developed to a considerable extent. However, there are some that cannot be developed or expressed under that condition but require a three-dimensional growth pattern. In this paper the authors explore the capacity of early to long-term passage airway epithelial cells (human and hamster) to resume architectures and functions existing in the native tissue in the specific environments given in vitro.
CC 4-1 (Toxicology)
IT Lung
(**epithelial** stem cell; in vitro reconstituted tissue as an alternative to human **respiratory** tract)
IT Bronchi
Lung
Respiratory tract
(**epithelium**; in vitro reconstituted tissue as an alternative to human **respiratory** tract)
IT **Air pollution**
Cell junction
Endoplasmic reticulum
Environmental pollution
Golgi apparatus
Respiratory tract
(in vitro reconstituted tissue as an alternative to human respiratory tract)
IT **Bioassay**
(in vitro tissue system; in vitro reconstituted tissue as an alternative to human respiratory tract)

L136 ANSWER 23 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:520177 CAPLUS

DOCUMENT NUMBER: 122:258315
TITLE: In vitro exposure of rabbit tracheal epithelium to SO2: Effects on morphology and **ciliary beating**
AUTHOR(S): Blanquart, C.; Giuliani, I.; Houcine, O.; Jeulin, C.; Guennou, C.; Marano, F.
CORPORATE SOURCE: Lab. Cytophysiologie Toxicologie Cellulaire, Universite Paris VII Denis Diderot, Paris, 75251, Fr.
SOURCE: Toxicology in Vitro (1995), 9(2), 123-32
CODEN: TIVIEQ; ISSN: 0887-2333
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 02 May 1995
AB The aim of this in vitro study was to characterize the direct effects of short-term exposure to low concns. of sulfur dioxide (SO2) on both the morphol. and the physiol. of rabbit tracheal primary cultures. SEM studies revealed that ciliated cells exposed for 1 h to 10 ppm or 30 ppm SO2 exhibited aggregated cilia. TEM revealed numerous swollen mitochondria in cells exposed to 30 ppm SO2 for 1 h. This morphol. damage to cells was coupled with physiol. alterations. A 25% decrease in **ciliary beat** frequency (CBF) was measured in cells exposed to 30 ppm SO2. This inhibition was partially reversible within 24 h. This SO2 concentration also induced a significant depletion of cellular ATP content which was completely restored after a 24-h recovery period. A correlation was found between cellular ATP level depletion and CBF decrease.
CC **4-3** (Toxicology)
Section cross-reference(s): **59**
ST tracheal epithelium sulfur dioxide morphol; **ciliary beating** tracheal epithelium sulfur dioxide
IT **Air pollution**
Cell morphology
(sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
IT Trachea (anatomical)
(epithelium, sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
IT Trachea (anatomical)
(**epithelium, ciliated** cell, sulfur trioxide effect on trachea **epithelium** with respect to **ciliary beating** and morphol.)
IT 7446-09-5, Sulfur dioxide, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
IT 7704-34-9, Sulfur, biological studies 14265-45-3, Sulfite 15181-46-1, Sulfite (HSO31-)
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)
IT 56-65-5, 5'-ATP, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(sulfur trioxide effect on trachea epithelium with respect to **ciliary beating** and morphol.)

L136 ANSWER 24 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN

DOCUMENT NUMBER: 115:2801
TITLE: Assessing the toxicity of environmental gas pollutants on **respiratory ciliated epithelia**
CORPORATE SOURCE: Universite de Paris V, Lab. Diffus. Inelastique Lumiere, Paris, Fr.
SOURCE: Report (1990), Order No. PB91-110841, 110 pp. Avail.: NTIS
From: Gov. Rep. Announce. Index (U. S.) 1991, 91(3), Abstr. No. 106,921
DOCUMENT TYPE: Report
LANGUAGE: French
ED Entered STN: 12 Jul 1991
AB The authors developed a relatively easy and reproducible technique for growing mammalian tracheal epithelial cultures, which they used to determine criteria for assessing the action or toxic mols. on epithelial cells, particularly ciliated cells. They designed a series of tests, including cilio-inhibition, viability, and proliferation-inhibition tests, for classifying the mols. tested. They also developed methods for measuring cell physiol. activity, including measurement of: (1) **ciliary beat** frequency through image anal. and phys. measurements; (2) proliferation through image anal. and fluorescent marking; and (3) viability through intracellular penetration of stains. This report discusses the methods developed, which can be used for any kind of mol., as well as the specific study of three reference mols. known for their toxicity to mammalian respiratory systems: acrolein, parathion, and caryololysine.
CC **4-3** (Toxicology)
Section cross-reference(s): 59
ST gas pollutant **respiratory** tract **epithelium**
IT **Air pollution**
(gas pollutants toxicity to **respiratory ciliated epithelium** in relation to)
IT **Respiratory** tract
(**epithelium, ciliated**, gas pollutants toxicity to)

L136 ANSWER 26 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1980:544183 CAPLUS
DOCUMENT NUMBER: 93:144183
TITLE: Response of **ciliated epithelium** to ozone and sulfuric acid
AUTHOR(S): Grose, Elaine C.; Gardner, Donald E.; Miller, Frederick J.
CORPORATE SOURCE: Health Effects Res., Northrop Serv., Inc., Research Triangle Park, NC, 27709, USA
SOURCE: Environmental Research (1980), 22(2), 377-85
CODEN: ENVRAL; ISSN: 0013-9351
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 12 May 1984
AB The effects of O3, H2SO4, and their interaction on ciliary activity were investigated. Following in vivo exposure to various concns. of O3 and H2SO4, ciliary activity of isolated tracheal ring cultures was microscopically determined under stroboscopic illumination. Assay of tracheal rings immediately after a 2-h exposure to 880 μg H2SO4/m3 showed a decrease in **ciliary beating** frequency from controls. Following 72 h in vitro maintenance, there was still a depression in ciliary activity of treatment cultures. In vivo recovery studies indicated that ciliary activity had returned to the normal range 72 h after exposure. Exposure to 196 μg O3/m3 for 3 h resulted in no significant difference from controls in ciliary activity. Expts. designed

ACCESSION NUMBER: 1992:77938 CAPLUS
DOCUMENT NUMBER: 116:77938
TITLE: Primary cultures of tracheal **epithelial** cells for the evaluation of **respiratory** toxicity
AUTHOR(S): Blanquart, C.; Romet, S.; Baeza, A.; Guennou, C.; Marano, F.
CORPORATE SOURCE: Lab. Cytophysiol. Toxicol. Cell., Univ. Paris 7, Paris, 75005, Fr.
SOURCE: Toxicology in Vitro (1991), 5(5-6), 499-502
CODEN: TIVIEQ; ISSN: 0887-2333
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 06 Mar 1992
AB Cytotoxic assays have been carried out to evaluate toxic effects of acrolein, mechlorethamine, parathion and paraoxon, using primary cultures of rabbit tracheal epithelium obtained by the explant outgrowth technique. The effect of drug exposure was first evaluated by the tetrazolium salt assay, neutral red uptake and release of cellular lactate dehydrogenase. These assays, previously developed for cell line cultures, were adapted to this model. In addition, the authors present an easy and rapid method to evaluate the effect of chems. on cell proliferation. This was performed using an image anal. method, and was the most sensitive marker to determine the acute toxicity of chems.
CC 4-1 (Toxicology)
Section cross-reference(s): 1
ST trachea **epithelium respiratory** toxicity evaluation; acrolein **respiratory** toxicity evaluation trachea **epithelium**; mechlorathamine **respiratory** toxicity evaluation trachea **epithelium**; parathion **respiratory** toxicity evaluation trachea **epithelium**; paraoxon **respiratory** toxicity evaluation trachea **epithelium**
IT **Respiratory** tract
(chemical toxicity to, evaluation of, by trachea **epithelium** toxicity assays)
IT Neoplasm inhibitors
(mechlorethamin as, toxicity of, to trachea **epithelium**, in **respiratory** toxicity evaluation)
IT **Toxicity**
(of chems., to trachea **epithelium**, in **respiratory** toxicity evaluation)
IT **Bioassay**
(of **respiratory** toxicity, in trachea **epithelium**)
IT Cell proliferation
(toxic chemical effect on, by trachea **epithelium**, in **respiratory** toxicity evaluation)
IT Cytotoxic agents
(toxicity of, to trachea **epithelium**, in **respiratory** toxicity evaluation)
IT Trachea (anatomical)
(**epithelium**, chemical toxicity to, in **respiratory** toxicity evaluation)
IT 51-75-2 56-38-2, Parathion 107-02-8, Acrolein, biological studies 311-45-5
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(toxicity of, to trachea **epithelium**, in **respiratory** toxicity evaluation)

L136 ANSWER 25 OF 47 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1991:402801 CAPLUS

to investigate the effects of a sequential exposure to O3 followed by H2SO4 indicated a decrease in the **ciliary beating** frequency of exposed animals which was less than that observed with H2SO4 alone. Thus, combined action expts. are extremely relevant in assessing the toxicity of environmental pollutants.

CC 4-3 (Toxicology)

IT **Air pollution**
(by ozone and sulfuric acid, ciliary activity in relation to)

L136 ANSWER 27 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005234517 EMBASE
TITLE: Hyperoxia-induced changes in human airway epithelial cells: The protective effect of perflubron.
AUTHOR: Babu P.B.R.; Chidekel A.; Shaffer T.H.
CORPORATE SOURCE: Dr. A. Chidekel, Department of Pediatrics, Alfred I. duPont Hospital for Children, 1600 Rockland Road, Wilmington, DE 19803, United States. achidek@nemours.org
SOURCE: Pediatric Critical Care Medicine, (2005) Vol. 6, No. 2, pp. 188-194.
Refs: 53
ISSN: 1529-7535
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20050616
Last Updated on STN: 20050616

ABSTRACT: Objective: To determine the protective effect of perflubron (PFB), a type of perfluorochemical liquid, in hyperoxia-induced cellular injury in the human airway epithelial cells. Design: A controlled, in vitro laboratory study. Setting: Tertiary-care children's hospital. Subjects: Human airway epithelial cells. Interventions: Human airway epithelial cells, Calu-3 cells, grown on polycarbonate porous filters at an air-liquid interface culture were exposed to normoxic (FiCO(2) = 5%, balance air) or hyperoxic (FIO(2) = 95%, balance CO(2)) conditions. Hyperoxia-induced cellular changes were monitored by measuring transepithelial resistance (TER) of monolayers, histology of cells, total protein, and interleukin-8 (IL-8) secretion in apical surface fluid (ASF) washings. Under hyperoxic conditions, the protective effect of PFB was assessed by directly adding PFB liquid to the apical surface of monolayers. Measurements and main results: During hyperoxic gas-liquid interface culture, Calu-3 monolayers exhibited a loss of cellular integrity morphologically, decreased protein concentration, and IL-8 level in ASF washings. During hyperoxic PFB-liquid interface culture, there was an overall increase in TER value of monolayers, improved histology, decreased total protein secretion in ASF washings, and unaltered IL-8 secretion. Cytomorphologic observations of PFB-treated Calu-3 cells indicated the presence of varying numbers of differently sized intracellular vacuoles during both normoxic and hyperoxic conditions. Conclusions: We conclude that the air-liquid interface culture of Calu-3 may be helpful in understanding mechanisms of lung injuries caused in clinical practice, and PFB protects against hyperoxia-induced airway epithelial cell injury by promoting cellular integrity as well as cytologic modifications. PFB-liquid interface culture of Calu-3 may be a useful in vitro model for

studying the cytoprotective role of liquid ventilation.

CONTROLLED TERM: Medical Descriptors:
*hyperoxia
*cell damage
***respiratory epithelium**
epithelium cell
in vitro study
laboratory test
pediatric hospital
cell growth
filter
air
liquid
cell culture
monolayer culture
histology
cytokine release
cell surface
gas
cell structure
protein secretion
cell count
cell vacuole
lung injury
cell protection
liquid ventilation
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*perfluorooctyl bromide: PD, pharmacology
polycarbonate
interleukin 8: EC, endogenous compound
CAS REGISTRY NO.: (perfluorooctyl bromide) 423-55-2; (polycarbonate) 24936-68-3, 25766-59-0; (interleukin 8) 114308-91-7

L136 ANSWER 28 OF 47 EMBASE

ACCESSION NUMBER: 2004464305 EMBASE
TITLE: Regulation of human airway ciliary beat frequency by intracellular pH.
AUTHOR: Sutto Z.; Conner G.E.; Salathe M.
CORPORATE SOURCE: M. Salathe, Div. of Pulmonary/Critical Care Med., Univ. of Miami School of Medicine, 1600 NW 10th Avenue, Miami, FL 33136, United States. msalathe@miami.edu
SOURCE: Journal of Physiology, (15 Oct 2004) Vol. 560, No. 2, pp. 519-532.
Refs: 68
ISSN: 0022-3751 CODEN: JPHYA7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20041119

Searched by Barb O'Bryen, STIC 2-2518

Last Updated on STN: 20041119

ABSTRACT: pH(i) affects a number of cellular functions, but the influence of pH(i) on mammalian ciliary beat frequency (CBF) is not known. CBF and pH(i) of single human tracheobronchial epithelial cells in submerged culture were measured simultaneously using video microscopy (for CBF) and epifluorescence microscopy with the pH-sensitive dye BCECF. Baseline CBF and pH(i) values in bicarbonate-free medium were 7.2 ± 0.2 Hz and 7.49 ± 0.02, respectively (n = 63). Alkalization by ammonium pre-pulse to pH(i) 7.78 ± 0.02 resulted in a 2.2 ± 0.1 Hz CBF increase (P < 0.05). Following removal of NH(4)Cl, pH(i) decreased to 7.24 ± 0.02 and CBF to 5.8 ± 0.1 Hz (P < 0.05). Removal of extracellular CO(2) to change pH(i) resulted in similar CBF changes. Pre-activation of cAMP-dependent protein kinase (10 μM forskolin), broad inhibition of protein kinases (100 μM H-7), inhibition of PKA (10 μM H-89), nor inhibition of phosphatases (10 μM cyclosporin + 1.5 μM okadaic acid) changed pH(i)-mediated changes in CBF, nor were they due to [Ca(2+)](i) changes. CBF of basolaterally permeabilized human trachcobronchial cells, re-differentiated at the air-liquid interface, was 3.9 ± 0.3, 5.7 ± 0.4, 7.0 ± 0.3 and 7.3 ± 0.3 Hz at basolateral i.e., intracellular pH of 6.8, 7.2, 7.6 and 8.0, respectively (n = 18). Thus, intracellular alkalization stimulates, while intracellular acidification attenuates human airway CBF. Since phosphorylation and [Ca(2+)](i) changes did not seem to mediate pH(i)-induced CBF changes, pH(i) may directly act on the ciliary motile machinery. .COPYRGT. The Physiological Society 2004.

CONTROLLED TERM:

Medical Descriptors:
***respiratory epithelium**
***ciliary motility**
*cell pH
cell function
mammal
tracheobronchial tree
epithelium cell
cell culture
microscopy
epifluorescence microscopy
culture medium
alkalinization
statistical analysis
enzyme inhibition
cell permeabilization
cell differentiation
air
liquid
surface property
basolateral membrane
acidification
human
controlled study
human tissue
human cell
article
priority journal
Drug Descriptors:
dye
bicarbonate
ammonia
carbon dioxide
cyclic AMP
protein kinase: EC, endogenous compound
forskolin

Searched by Barb O'Bryen, STIC 2-2518

phosphatase: EC, endogenous compound
cyclosporin
okadaic acid

CAS REGISTRY NO.: (bicarbonate) 144-55-8, 71-52-3; (ammonia) 14798-03-9, 51847-23-5, 7664-41-7; (carbon dioxide) 124-38-9, 58561-67-4; (cyclic AMP) 60-92-4; (protein kinase) 9026-43-1; (forskolin) 66575-29-9; (phosphatase) 9013-05-2; (cyclosporin) 79217-60-0; (okadaic acid) 78111-17-8

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ACCESSION NUMBER: 2004381036 EMBASE

TITLE: Exposure of differentiated airway epithelial cells to volatile smoke in vitro.

AUTHOR: Beisswenger C.; Platz J.; Seifart C.; Vogelmeier C.; Bals R.

CORPORATE SOURCE: Dr. R. Bals, Department of Internal Medicine, Division of Pulmonology, Hosp. of the University of Marburg, Baldingerstrasse 1, DE-35043 Marburg, Germany. bals@mailer.uni-marburg.de

SOURCE: Respiration, (2004) Vol. 71, No. 4, pp. 402-409. Refs: 27
ISSN: 0025-7931 CODEN: RESPBD

COUNTRY: Switzerland

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20040924
Last Updated on STN: 20040924

ABSTRACT: Background: Cigarette smoke (CS) is the predominant pathogenetic factor in the development of chronic bronchitis and chronic obstructive pulmonary disease. The knowledge about the cellular and molecular mechanisms underlying the smoke-induced inflammation in epithelial cells is limited. Objectives: The aim of this study was to develop an in vitro model to monitor the effects of volatile CS on differentiated airway epithelial cells. Methods: The airway epithelial cell line MM-39 and primary human bronchial epithelial cells were cultivated as air-liquid interface cultures and exposed directly to volatile CS. We used two types of exposure models, one using ambient air, the other using humidified and warm air. Cytokine levels were measured by quantitative PCR and ELISA. Phosphorylation of p38 MAP kinase was assessed by Western blot analysis. To reduce the smoke-induced inflammation, antisense oligonucleotides directed against the p65 subunit of NF-κB were applied. Results: Exposure of epithelia to cold and dry air resulted in a significant inflammatory response. In contrast, exposure to humidified warm air did not elicit a cellular response. Stimulation with CS resulted in upregulation of mRNA for IL-6 and IL-8 and protein release. Exposure to CS combined with heat-inactivated bacteria synergistically increased levels of the cytokines. Reactions of differentiated epithelial cells to smoke are mediated by the MAP kinase p38 and the transcription factor NF-κB. Conclusions: We developed an exposure model to examine the consequences of direct exposure of differentiated airway epithelial cells to volatile CS. The model enables to measure the cellular reactions to smoke exposure and to determine the outcome of therapeutic interventions. Copyright .COPYRGT. 2004 S. Karger AG, Basel.

CONTROLLED TERM: Medical Descriptors:
***smoke**
*chronic obstructive lung disease: ET, etiology
respiratory epithelium

Searched by Barb O'Bryen, STIC 2-2518

cell differentiation
disease model
cell culture
ambient air
humidity
enzyme linked immunosorbent assay
quantitative analysis
polymerase chain reaction
inflammation
enzyme phosphorylation
upregulation
protein secretion
human
controlled study
human cell
article
priority journal
Drug Descriptors:
*volatile agent
cytokine: EC, endogenous compound
mitogen activated protein kinase: EC, endogenous compound
immunoglobulin enhancer binding protein: EC, endogenous compound
interleukin 6: EC, endogenous compound
interleukin 8: EC, endogenous compound
CAS REGISTRY NO.: (mitogen activated protein kinase) 142243-02-5; (interleukin 8) 114308-91-7

L136 ANSWER 30 OF 47 EMBASE COPYRIGHT (c) 2005 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002341737 EMBASE
TITLE: Effects of hexamethylene diisocyanate exposure on human airway epithelial cells: In vitro cellular and molecular studies.
AUTHOR: Wisnewski A.V.; Liu Q.; Miller J.-J.; Magoski N.; Redlich C.A.
CORPORATE SOURCE: A.V. Wisnewski, Yale University School of Medicine, Department of Internal Medicine, 333 Cedar Street, New Haven, CT 06520, United States. adam.wisnewski@yale.edu
SOURCE: Environmental Health Perspectives, (2002) Vol. 110, No. 9, pp. 901-907.
Refs: 35
ISSN: 0091-6765 CODEN: EVHPAZ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
029 Clinical Biochemistry
046 Environmental Health and Pollution Control
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20021017
Last Updated on STN: 20021017

ABSTRACT: In this study we developed an in vitro exposure model to investigate the effects of hexamethylene diisocyanate (HDI) on human airway epithelial cells at the cellular and molecular level. We used immunofluorescence analysis (IFA) to visualize the binding and uptake of HDI by airway epithelial cell lines (A549 and NCI-NCI-H292) and microarray technology to identify HDI sensitive genes. By IFA, we observed that subcytotoxic concentrations of HDI form microscopic micelles that appear to be taken up by cells over a 3-hr

period postexposure. Microarray analysis (4.6K genes) of parallel cultures identified four genes (thioredoxin reductase, dihydrodiol dehydrogenase, TG interacting factor, and stanniocalcin) whose mRNA levels were up-regulated after HDI exposure. Northern analysis was used to confirm that HDI increased message levels of these four genes and to further explore the dose dependence and kinetics of the response. The finding that HDI exposure increases thioredoxin reductase expression supports previous studies suggesting that HDI alters thiol-redox homeostasis, an important sensor of cellular stress. Another of the HDI-increased genes, a dihydrodiol dehydrogenase, encodes a protein previously shown to be specifically susceptible to HDI conjugation, and known to detoxify other hydrocarbons. Together, the data describe a novel approach for investigating the effects of HDI binding and uptake by human airway epithelial cells and begin to identify genes that may be involved in the acute response to exposure.

CONTROLLED TERM: Medical Descriptors:
environmental exposure
respiratory epithelium
epithelium cell
in vitro study
immunofluorescence test
binding kinetics
transport kinetics
cell line
DNA microarray
gene identification
cytotoxicity
concentration (parameters)
microscopy
micelle
cell culture
Northern blotting
gene expression
oxidation reduction state
stress
human
controlled study
human cell
article
nucleotide sequence
priority journal
Drug Descriptors:
*hexamethylene diisocyanate: TO, drug toxicity
DNA: EC, endogenous compound
thioredoxin reductase: EC, endogenous compound
dihydrodiol dehydrogenase: EC, endogenous compound
hypocalcin: EC, endogenous compound
messenger RNA: EC, endogenous compound

CAS REGISTRY NO.: (hexamethylene diisocyanate) 11142-52-2, 822-06-0; (DNA) 9007-49-2; (thioredoxin reductase) 9074-14-0; (dihydrodiol dehydrogenase) 37255-32-6; (hypocalcin) 76687-96-2

GENE NUMBER: GENBANK AA085318 referred number; GENBANK AA453335 referred number; GENBANK R83270 referred number; GENBANK R93124 referred number

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ACCESSION NUMBER: 2001001548 EMBASE

TITLE: Coal fly ash and mineral dust for toxicology and particle characterization studies: Equipment and methods for PM2.5-

and PM1-enriched samples.

AUTHOR: Veranth J.M.; Smith K.R.; Aust A.E.; Dansie S.L.; Griffin J.B.; Hu A.A.; Huggins M.L.; Lighty J.S.

CORPORATE SOURCE: J.M. Veranth, Dept. of Chemical/Fuels Engineering, University of utah, 207 KRC 1495 East 100 South, Salt Lake City, UT 84112-1114, United States

SOURCE: Aerosol Science and Technology, (2000) Vol. 32, No. 2, pp. 127-141.
Refs: 39
ISSN: 0278-6826 CODEN: ASTYDQ

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 046 Environmental Health and Pollution Control
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 20010111
Last Updated on STN: 20010111

ABSTRACT: Laboratory methods to produce particle samples from known, reproducible sources with sufficient mass to perform both detailed characterization and replicated in vitro toxicological assays are described. These samples are being used to study the ability of inhalable particles to produce abnormal concentrations of intracellular iron, resulting in the production of reactive oxygen species in cultured airway epithelial cells. Bulk samples of size fractionated particles from laboratory-generated coal fly ash and from simulated fugitive mining tailings and road dust were collected as surrogates for important sources of iron-bearing particles in the ambient air. An Andersen cascade impactor was used to produce particle samples enriched in three size ranges: >10 μm, 10-2.5 μm, and <2.5 μm aerodynamic diameter. A multijet preseparator and rectangular slot virtual impactor were used to produce a fraction enriched in particles below 1 μm. Data on the particle production conditions, production rates, and particle sample quality are provided to illustrate the feasibility of the experimental approach. The amount of iron mobilized from particles by a physiologically-relevant chelator does not correlate with the total iron. This supports the hypothesis that particle characteristics and iron speciation are important for the production of abnormal iron concentrations in cultured type A549 human airway epithelial cells. Comparison of results obtained with these surrogate particles to previous work with urban particulate standard reference materials (SRM 1648 and SRM 1649) suggests particle sources and size fractions that should be emphasized for detailed characterization of particle morphology and mineralogy.

CONTROLLED TERM: Medical Descriptors:
*fly ash
*mineral dust
*toxicology
particle size
bioaccumulation
cell culture
aerosol
in vitro study
inhalation
respiratory epithelium
simulation
mining
ambient air
feasibility study
hypothesis
human
controlled study
human cell

Searched by Barb O'Bryen, STIC 2-2518

article
priority journal
Drug Descriptors:
*coal
*mineral
iron
iron chelate
CAS REGISTRY NO.: (iron) 14093-02-8, 53858-86-9, 7439-89-6

L136 ANSWER 32 OF 47 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 8

ACCESSION NUMBER: 1995-0499318 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Tracheal epithelial cells in vitro as a model to study genotoxicity of **airborne** particulates
AUTHOR: HORNBERG C.; SEEMAYER N. H.
REINHARDT Christoph A. (ed.); BENFORD Diane J. (ed.); BLAAUBOER Bas J. (ed.)
CORPORATE SOURCE: Medical inst. environmental hyg., Heinrich-Heine-Univ. 40223 Duesseldorf, Germany, Federal Republic of
Swiss inst. alternatives animal testing, 8005 Zuerich, Switzerland
SOURCE: Toxicology in vitro, (1995), 9(4), 397-402 [5 p.], 30 refs.
Conference: 8 INVITOX 94 : International wokshop on in vitro toxicology, Kartause Ittingen (Switzerland), 20 Sep 1994
ISSN: 0887-2333 CODEN: TIVIEQ
DOCUMENT TYPE: Journal; Conference
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-21203, 354000054146370070
ABSTRACT: The major target site of **airborne** particulates is the tracheobronchial **epithelium** of the **respiratory** tract. It is also the origin of the most common cancer in man, bronchogenic carcinoma. Rodent tracheal epithelial cells in **culture** can be used to study the genotoxic activity of **airborne** particulates leading to mutation and cancer. **Airborne** particulates were collected in the heavily industrialized Rhine-Ruhr region using a high volume sampler HVS 150 (Stroehlein Instruments) equipped with glass fibre filters. Chemical substances were extracted with di-chloromethane or methanol and quantitatively transferred to dimethyl sulfoxide for tissue **culture** experiments. Tracheal epithelial cells of the Syrian golden hamster and the Wistar rat were dissociated by pronase treatment and cultivated in a 'complex' medium. The induction of sister chromatid exchanges was used as a sensitive **bioassay** for detection of genotoxic activity of **airborne** particulates. Extracts of **airborne** particulateds led to a dose-related highly significant induction of sister chromatid exchanges in cell **cultures** of tracheal epithelial cells of the hamster and the rat. Even quantities of chemical substances equivalent to

airborne particulates from less than 1 m.sup.3 air were markedly genotoxic.

CLASSIFICATION CODE: 002B03N; Life sciences; Medical sciences; Toxicology
CONTROLLED TERM: Suspended particle; Toxicity; Carcinogen; Carcinogenicity testing; Investigation method; Epithelial cell; Trachea; Dose activity relation; DNA; Sister chromatid exchange; Chromosomal aberration; Mutagenicity testing

BROADER TERM: Respiratory disease

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ACCESSION NUMBER: 2000-0063051 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2000 INIST-CNRS. All rights reserved.

TITLE (IN ENGLISH): Effects of aqueous extracts of PM.sub.1.sub.0 filters from the Utah Valley on human airway epithelial cells

AUTHOR: FRAMPTON M. W.; GHIO A. J.; SAMET J. M.; CARSON J. L.; CARTER J. D.; DEVLIN R. B.

CORPORATE SOURCE: Department of Medicine and Environmental Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York 14642, United States; Human Studies Division, United States Environmental Protection Agency, Chapel Hill, North Carolina 27599, United States; Center for Environmental Medicine and Lung Biology, University of North Carolina, Chapel Hill, North Carolina 27599, United States

SOURCE: American journal of physiology. Lung cellular and molecular physiology, (1999), 21(5), L960-L967, 31 refs.
ISSN: 1040-0605

DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-22200, 354000080406110140

ABSTRACT: We hypothesized that the reduction in hospital respiratory admissions in the Utah Valley during closure of a local steel mill in 1986-1987 was attributable in part to decreased toxicity of ambient air particles. **Sampling** filters for particulate matter < 10 μm (PM.sub.1.sub.0) were obtained from a Utah Valley monitoring station for the year before (year 1), during (year 2), and after (year 3) the steel mill closure. Aqueous extracts of the filters were analyzed for metal content and oxidant production and added to **cultures** of human **respiratory epithelial** (BEAS-2B) cells for 2 or 24 h. Year 2 dust contained the lowest concentrations of soluble iron, copper, and zinc and showed the least oxidant generation. Only dust from year 3 caused cytotoxicity (by microscopy and lactate dehydrogenase release) at 500 μg/ml. Year 1 and year 3, but not year 2, dust induced expression of interleukin-6 and -8 in a dose-response fashion. The effects of ambient air particles on human respiratory epithelial cells vary significantly with time and metal concentrations.

CLASSIFICATION CODE: 002B03L05; Life sciences; Medical sciences; Toxicology
CONTROLLED TERM: Heavy metal; Pollutant; Toxicity; Epithelial cell;

Searched by Barb O'Bryen, STIC 2-2518

Respiratory tract; Interleukin 8; Interleukin 6; Aqueous solution; Extract; Filter; Human; Utah

BROADER TERM: **Air pollution**; Respiratory system; United States; North America; America

L136 ANSWER 34 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V.
on STN DUPLICATE

ACCESSION NUMBER: 1994080637 ESBIOBASE

TITLE: In vitro exposure of tracheobronchial epithelial cells and of tracheal explants to ozone

AUTHOR: Tarkington B.K.; Wu R.; Sun W.-M.; Nikula K.J.; Wilson D.W.; Last J.A.

CORPORATE SOURCE: J.A. Last, Calif. Regional Primate Res. Center, University of California, Davis, CA 95616-8542, United States.

SOURCE: Toxicology, (1994), 88/1-3 (51-68)
CODEN: TXCYAC ISSN: 0300-483X

DOCUMENT TYPE: Journal; Article

COUNTRY: Ireland

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: An in vitro system for exposing **respiratory epithelial** cells or explant tissues to ozone has been developed and characterized. This system is designed to generate and monitor consistent, reproducible levels of ozone, over a range of concentrations, in a humidified atmosphere, and to allow an exposure time of 24 h or longer. Based on chemical analysis, highly reproducible concentrations of ozone are delivered throughout the chamber, with a coefficient of variation of < 5% between five replicate vials exposed to 0.5 ppm of ozone for 50 min. The viability of **cultured** human tracheobronchial epithelial cells, as measured by the ability to oxidize a vital dye, and of rat tracheal epithelium, as measured by total numbers of necrotic cells in tracheal explants, after ozone exposure was examined in this system. Responses of **cultured** cells to ozone exposure as measured by **bioassay** were consistent with the observed low level of variability of ozone concentration between replicate incubation dishes or vials. Responses of **cultured** cells to ozone were proportional to duration of exposure and inversely proportional to the volume of medium covering the cells. We conclude that this newly developed in vitro exposure system will allow relatively simple and convenient exposure of **cultured** cells or organs to ozone or other gaseous agents under highly controlled and reproducible conditions.

CLASSIFICATION CODE: 90.10.3 TOXICOLOGY: METHODS IN CLINICAL AND EXPERIMENTAL TOXICOLOGY: Toxicity Tests
90.5.1.2 TOXICOLOGY: EXPERIMENTAL TOXICOLOGY (by agent): Industrial and **Environmental Pollutants**: Other inorganic
90.6.6 TOXICOLOGY: CLINICAL AND EXPERIMENTAL TOXICOLOGY (by target organ): Respiratory

SUPPLEMENTARY TERM: Ozone; Cell **culture**; **Air pollution**

Searched by Barb O'Bryen, STIC 2-2518

L136 ANSWER 35 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1999279528 ESBIOBASE
TITLE: Effects of aqueous extracts of PM.sub.1.sub.0 filters from the Utah Valley on human airway epithelial cells
AUTHOR: Frampton M.W.; Ghio A.J.; Samet J.M.; Carson J.L.; Carter J.D.; Devlin R.B.
CORPORATE SOURCE: M.W. Frampton, Pulmonary and Critical Care Unit, University of Rochester, School of Medicine/Dentistry, 601 Elmwood Ave., Rochester, NY 14642-8692, United States.
SOURCE: American Journal of Physiology - Lung Cellular and Molecular Physiology, (1999), 277/5 21-5 (L960-L967), 31 reference(s)
CODEN: APLPE7 ISSN: 1040-0605
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT: We hypothesized that the reduction in hospital respiratory admissions in the Utah Valley during closure of a local steel mill in 1986-1957 was attributable in part to decreased toxicity of ambient air particles. **Sampling** filters for particulate matter < 10 μm (PM.sub.1.sub.0) were obtained from a Utah Valley monitoring station for the year before (year 1), during (year 2), and after (year 3) the steel mill closure. Aqueous extracts of the filters were analyzed for metal content and oxidant production and added to **cultures** of human **respiratory epithelial** (BEAS-2B) cells for 2 or 24 h. Year 2 dust contained the lowest concentrations of soluble iron, copper, and zinc and showed the least oxidant generation. Only dust from year 3 caused cytotoxicity (by microscopy and lactate dehydrogenase release) at 500 μg/ml. Year 1 and year 3, but not year 2, dust induced expression of interleukin-6 and -8 in a dose-response fashion. The effects of ambient air particles on human respiratory epithelial cells vary significantly with time and metal concentrations.
CLASSIFICATION CODE: 89.5.1.7 CELL AND DEVELOPMENTAL BIOLOGY: CELL TYPES AND BIOLOGY: Cell Types: Epithelial and endothelial cells
SUPPLEMENTARY TERM: **Air pollution**; Metals; Interleukin-6; Interleukin-8; Toxicity

L136 ANSWER 36 OF 47 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1999251260 ESBIOBASE
TITLE: Intranasal toxicity of BMS-181885, a novel 5-HT1 agonist
AUTHOR: Schulze G.E.; Proctor J.E.; Dominick M.A.; Weiss A.E.; Flint O.P.; Srinivas N.R.; Durham S.K.; Schilling B.E.
CORPORATE SOURCE: Dr. G.E. Schulze, Bristol-Myers Squibb Co., 6000 Thompson Road, Syracuse, NY 13057, United States. E-mail: schulzeg@bms.com
SOURCE: International Journal of Toxicology, (1999), 18/5 (285-296), 48 reference(s)
CODEN: IJTOFN ISSN: 1091-5818

Searched by Barb O'Bryen, STIC 2-2518

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
ABSTRACT: One-month intranasal toxicity studies were conducted with BMS-181885 at doses of 1.5, 9, or 15 mg/animal/day in rats and 4, 24, or 40 mg/animal/day in monkeys. A 1-month intermittent intranasal toxicity study was also conducted in monkeys at doses of 3, 6, and 12 mg/animal 3 days per week. BMS- 181885 was generally well tolerated in rats but resulted in dose-dependent nasal mucosal injury, primarily characterized by subacute inflammation of the nasal mucosa, and degeneration, single-cell necrosis, and/or erosion of the olfactory epithelium and, to a lesser extent, the **respiratory epithelium** . In monkeys, daily BMS-181885 administration was well tolerated and produced similar dose-dependent nasal injury primarily characterized by subacute inflammation of the nasal mucosa with degeneration and erosion of the olfactory epithelium. In a separate experiment, intermittent administration also resulted in dose-dependent nasal injury. In **cultured** rat nasal mucosal cells, BMS-181885 was toxic to olfactory epithelial cells with a range of mean IC.sub.5.sub.0s between 44 and 291 μM. In contrast, BMS-181885 had no effect on **respiratory epithelial** cells up to its maximum solubility. Cytochrome P450 inhibition had no effect on the toxicity of BMS-181885 in olfactory epithelial cells but produced dose-dependent toxicity in **respiratory epithelial** cells, which was not present previously. The in vitro data suggest that parent drug, rather than a toxic metabolite, caused the drug-associated nasal mucosal injury.
CLASSIFICATION CODE: 90.4.1.3 TOXICOLOGY: CLINICAL TOXICOLOGY (by agent): Industrial and **Environmental Pollutants**: Pesticides and agricultural chemicals
90.6.1 TOXICOLOGY: CLINICAL AND EXPERIMENTAL TOXICOLOGY (by target organ): Neuro-**sensory**
90.10.3 TOXICOLOGY: METHODS IN CLINICAL AND EXPERIMENTAL TOXICOLOGY: Toxicity Tests
SUPPLEMENTARY TERM: 5-HT agonist; Intranasal toxicity; In vitro; In vivo; Metabolism; Monkeys; Nasal toxicity; P450; Rats; Safety assessment; Serotonin

L136 ANSWER 37 OF 47 NIOSHTIC on STN
ACCESSION NUMBER: 1997:180032 NIOSHTIC
DOCUMENT NUMBER: NIOSH-00210743
TITLE: Toxic Effects of Sulfur Mustard on **Respiratory Epithelial** Cells in **Culture**
AUTHOR(S): Chevillard, M.; Lainee, P.; Robineau, P.; Puchelle, E.
SOURCE: Cell Biology and Toxicology, Vol. 8, No. 2, pages 171-181, 24 references
CODEN: CBTOE2
PUBLICATION DATE: Apr 1992
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH
ABSTRACT:

Searched by Barb O'Bryen, STIC 2-2518

The toxicity of sulfur-mustard (505-60-2) (SM) to respiratory epithelial cells was studied in-vitro. Respiratory epithelial cells isolated from male New-Zealand-white-rabbit tracheas were cultured and incubated with 0.1, 1, or 10 millimolar (mM) SM for 2 minutes. The effects on **ciliary** ***beat*** frequency were determined 30, 60, or 90 minutes and 2, 3, 4, 5, or 6 hours after treatment using a videomicroscopic technique. The cultures were observed for morphological changes by optical and electron microscopy. Control cultures developed a layer of adherent epithelial cells that progressively increased to form an outgrowth of differentiated and undifferentiated cells. The 1 and 10mM SM exposures arrested **ciliary beating** within 30 and 10 minutes, respectively. The 0.1mM exposure caused an irreversible arrest of **ciliary beating** in the outgrowth zone after 90 minutes in two of six cultures. **Ciliary beating** frequency was not affected by 0.1mM SM in the other four cultures. Morphological changes were assessed only in cultures treated with 0.1mM SM because of the less drastic effect on **ciliary beating** frequency. SM induced vacuolization of the ciliated cells after 1 hour. The outgrowth zone became less adherent after 2 hours and tended to roll up after 4 hours. The outgrowth zone eventually became completely detached. Treatment related ultrastructural changes included mitochondrial swelling that appeared after 1 hour and cytoplasmic abnormalities such as increases in the number of intermediate filaments and the development of organelle free zones near the cell periphery that appeared at 6 hours. The authors conclude that SM profoundly affects the viability of **respiratory epithelial** cell **cultures** . The sudden arrest of **ciliary beating** more likely reflects the death of ciliated respiratory epithelial cells than a specific ciliotoxic effect of SM.

CONTROLLED TERM: Mustard gas; Mammalian cells; In vitro studies; Cytotoxic effects; Dose response; Cellular function; Ultrastructure; Histomorphology; **Chemical warfare** agents

CAS REGISTRY NO.: 505-60-2 (sulfur-mustard)

L136 ANSWER 38 OF 47 INSPEC (C) 2005 IEE on STN

ACCESSION NUMBER: 2000:6665594 INSPEC

DOCUMENT NUMBER: A2000-18-8760F-022; B2000-09-7510J-031

TITLE: Homodyne mixing of scattered light as a novel technique for the measurement of **ciliary beat** frequency.

AUTHOR: Wilson, M.J. (Dept. of Med. Phys., Univ. Hosp., Birmingham, UK); Drake-Lee, A.; Wang, R.

SOURCE: Proceedings of the SPIE - The International Society for Optical Engineering (2000) vol.3915, p.170-7. 20 refs.
Published by: SPIE-Int. Soc. Opt. Eng
Price: CCCC 0277-786X/2000/$15.00
CODEN: PSISDG ISSN: 0277-786X .
SICI: 0277-786X(2000)3915L.170:HMSL;1-Q
Conference: Coherence Domain Optical Methods in Biomedical Science and Clinical Applications IV. San Jose, CA, USA, 24-26 Jan 2000
Sponsor(s): SPIE; IBOS Int. Biomed. Opt. Soc

DOCUMENT TYPE: Conference Article; Journal

TREATMENT CODE: Experimental

COUNTRY: United States

LANGUAGE: English

ABSTRACT: Cilia are finger like organelles present throughout the **epithelium** of the human **respiratory** tract. Typically 5-6 mu m long,

with a density of around 8/ mu m2, they 'beat' at frequencies of 10-20 Hz and propel an overlying mucus layer. The mucus traps **airborne** particulates providing an essential respiratory cleaning/infection control mechanism. A novel method for measurement of **ciliary beat** frequency in-vivo is presented. Homodyne mixing of scattered coherent light promises a relatively large signal with minimal sensitivity to vibrational when compared with existing methods: Vibrational susceptibility is minimized since all scattered pathlengths are similarly affected, while phase discrepancies introduced by the muco-ciliary surface produce high contrast speckle giving good signal quality. The need to illuminate a relatively small area of epithelium to give large speckle has been addressed using a 'shortened' gradient index rod as a combined delivery/focusing device. Results are good when the ciliary surface reflectivity is increased. However the relatively low reflection coefficient of the unprepared surface allows homodyne mixing from deeply scattered light giving a speckle structure too small to resolve and consequent signal loss. Evidence is presented that demonstrates how a source with limited temporal coherence might be used to suppress the interference from these 'longer pathlength' photons.

CLASSIFICATION CODE: A8760F Optical and laser radiation (medical uses); A8725D Biological transport; cellular and subcellular transmembrane physics; A4225F Optical diffraction and scattering; A8750B Interactions of biosystems with radiations; A8770E Patient diagnostic methods and instrumentation; A4225H Optical interference and speckle; A0760L Optical interferometry; A4225K Optical coherence; A4281P Fibre optic sensors; fibre gyros; B7510J Optical and laser radiation (biomedical imaging/measurement); B7230E Fibre optic sensors

CONTROLLED TERM: CELL MOTILITY; FIBRE OPTIC **SENSORS**; LASER APPLICATIONS IN MEDICINE; LIGHT COHERENCE; LIGHT SCATTERING; SPECKLE

SUPPLEMENTARY TERM: finger like organelles; homodyne mixing; phase discrepancies; scattered coherent light; epithelium; human respiratory tract; overlying mucus layer; **airborne particulates**; essential respiratory cleaning/infection control mechanism; **ciliary beat frequency in-vivo**; vibrational susceptibility; scattered pathlength; muco-ciliary surface; signal quality; shortened gradient index rod; combined delivery/focusing device; ciliary surface reflectivity; reflection coefficient; unprepared surface; deeply scattered light; speckle structure; signal loss; limited temporal coherence; longer pathlength photons

L136 ANSWER 39 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 2

ACCESSION NUMBER: 2001:311226 BIOSIS

DOCUMENT NUMBER: PREV200100311226

TITLE: Genotoxicity of nitroso compounds and sodium dichromate in a model combining organ cultures of human nasal epithelia and the comet assay.

Searched by Barb O'Bryen, STIC 2-2518

AUTHOR(S): Kleinsasser, Norbert H. [Reprint author]; Gamarra, Fernando; Bergner, Albrecht; Wallner, Barbara C.; Harreus, Ulrich A.; Juchhoff, Jutta; Kastenbauer, Ernst R.; Huber, Rudolf M.

CORPORATE SOURCE: Klinisch Experimentelle Onkologie, Klinik und Poliklinik fuer Hals-, Nasen- und Ohrenkranke, Ludwig-Maximilians-Universitaet Muenchen, Pettenkoferstrasse 4a, D-80336, Muenchen, Germany
norbert.kleinsasser@hno.med.uni-muenchen.de

SOURCE: ORL (Basel), (May-June, 2001) Vol. 63, No. 3, pp. 141-147. print.
CODEN: ORLJAH. ISSN: 0301-1569.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002

ABSTRACT:Genotoxic effects of xenobiotics are a possible step in tumor initiation in the mucosa of the upper aerodigestive tract. Using the comet assay, detecting genotoxicity in human tissue has been restricted to single incubations in vitro, but in vivo most xenobiotics harm their target in a repetitive or chronic manner. Therefore, we propose a model, which provides repetitive incubations in human upper aerodigestive tract mucosa cultures. ***Samples*** of human inferior nasal turbinate mucosa (n=25) were cultured according to a modified version of a technique originally described by Steinsvag. On day 1 fresh **samples** and on days 7, 9 and 11 organ cultures were incubated with N-nitrosodiethylamine (NDEA), sodium dichromate (Na2Cr2O7) and N'-methyl-N-nitro-N-nitroso-guanidine (MNNG). Mucosa ***samples*** and organ cultures, respectively, underwent a modified comet assay on days 1, 7 and 11. Genotoxicity could be shown for NDEA, Na2Cr2O7 and MNNG on days 1, 7 and 11. Duration of tissue culture and repetitive incubations did not significantly influence the results for NDEA. Nevertheless, Na2Cr2O7 and MNNG caused higher genotoxic effects on cultures subjected to the comet assay on day 11. This model may help to assess genotoxic hazards posed by **environmental pollutants** that have a cumulative character in repetitive or chronic exposure in vivo.

CONCEPT CODE:
Digestive system - Physiology and biochemistry 14004
Genetics - General 03502
Genetics - Human 03508
Respiratory system - Physiology and biochemistry 16004
Toxicology - General and methods 22501

INDEX TERMS:
Major Concepts
Genetics; Toxicology

INDEX TERMS:
Parts, Structures, & Systems of Organisms
aerodigestive tract: digestive system; nasal **epithelia**: **respiratory** system, organ **culture**

INDEX TERMS:
Chemicals & Biochemicals
nitroso compounds: genotoxicity; sodium dichromate: genotoxicity

INDEX TERMS:
Methods & Equipment
comet assay: toxicity testing method

ORGANISM:
Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

Searched by Barb O'Bryen, STIC 2-2518

REGISTRY NUMBER: 10588-01-9 (sodium dichromate)

L136 ANSWER 40 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 6

ACCESSION NUMBER: 1998:40981 BIOSIS
DOCUMENT NUMBER: PREV199800040981
TITLE: Comparative analysis of cyto- and genotoxic effects of **airborne** particulates on human and rodent respiratory cells in vitro.
AUTHOR(S): Hornberg, C.; Maciuleviciute, L.; Seemayer, N. H.
CORPORATE SOURCE: Med. Inst. Environ. Hyg., Univ. Duesseldorf, Auf m Hennekamp 50, D-40225 Duesseldorf, Germany
SOURCE: Toxicology In Vitro, (Oct., 1997) Vol. 11, No. 5, pp. 711-715. print.
CODEN: TIVIEQ. ISSN: 0887-2333.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 1998
Last Updated on STN: 14 Jan 1998

ABSTRACT: In our highly industrialized world **air pollution** has become an important topic. Beside gaseous pollutants **airborne** particulates are of great medical concern, containing several hundred mostly organic substances. They are incriminated to cause an excess mortality. ***Airborne*** particulates were collected in the heavily industrialized Ruhr region utilizing a high volume sampler HVS 150 (Strohlein Instruments) equipped with glass fibre filters. Chemical substances were extracted with dichloromethane and quantitatively transferred to dimethyl sulfoxide for tissue ***culture*** experiments. Cytotoxicity of extracts was determined by reduction of 'plating efficiency' of human cell line A-549 (pneumocyte type II). The induction of 'sister chromatid exchanges' was used as a sensitive ***bioassay*** for detection of genotoxic activity of **airborne** particulates. As target cells we utilized tracheal epithelial cells of the Syrian golden hamster and the rat, human bronchial epithelial cells of line BEAS-2B and human lymphocytes. Quantities of substances equivalent to ***airborne*** particulates from 4 and more ml air exerted cytotoxic effects, while quantities of substances from 0.5 ml of air were markedly genotoxic.

CONCEPT CODE:
Toxicology - General and methods 22501
Cytology - Animal 02506
Genetics - Animal 03506
Respiratory system - Physiology and biochemistry 16004
In vitro cellular and subcellular studies 32600
Respiratory system - General and methods 16001

INDEX TERMS:
Major Concepts
Toxicology

INDEX TERMS:
Parts, Structures, & Systems of Organisms
tracheal **epithelial** cells: **respiratory** system

INDEX TERMS:
Miscellaneous Descriptors
airborne particulates; cytotoxic effect; genotoxic effect; sister chromatid exchanges

ORGANISM:
Classifier
Cricetidae 86310
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Syrian golden hamster
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

ORGANISM:
Classifier

Searched by Barb O'Bryen, STIC 2-2518

Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
A-549: human pneumocytes
BEAS-2B: human bronchial epithelial cells
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

ORGANISM:

Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rat
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

L136 ANSWER 41 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:186433 BIOSIS
DOCUMENT NUMBER: PREV200500186977
TITLE: A new approach to **pharmacological** effects on **ciliary beat** frequency in cell cultures-exemplary measurements under Pelargonium sidoides extract.(EPs 7630).
AUTHOR(S): Neugebauer, P. [Reprint Author]; Mickenhagen, A.; Siefer, O.; Walger, M.
CORPORATE SOURCE: ENT Dept, Univ Cologne, Cologne, Germany
hno.neugebauer@uni-koeln.de
SOURCE: Phytomedicine (Jena), (January 2005) Vol. 12, No. 1-2, pp. 46-51. print.
ISSN: 0944-7113 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 May 2005
Last Updated on STN: 18 May 2005

ABSTRACT:The **ciliary beat** frequency (CBF) is an important parameter of the defence mechanism of the mucociliary system. We present a new method to determine **pharmacological** effects on CBF in vitro. Ciliated cell cultures of human nasal epithelium were obtained from partial resection of hyperplastic inferior turbinates in rhinosurgery. An adherent monolayer culture of ciliated cells was present after 10 days in vitro. This study exemplary examines, if a special extract from the roots of Pelargonium sidoides (EPs 7630) has an effect on the CBF in vitro. The influence of three concentrations of the extract (1, 30, 100 mug/ml) was tested. EPs 7630 significantly and concentration-dependently increased CBF to 123% at 30 mug/ml and to 133% at 100 mug/ml compared to the equilibration phase (100%). After rinsing with extract-free medium the CBF of cultured cells returned to nearly the normal range. In future, drug manipulation of the CBF by local application of rhinologics could be a new therapeutical concept in the treatment of upper airway diseases.

CONCEPT CODE:
Pathology - Therapy 12512
Respiratory system - Physiology and biochemistry 16004
Respiratory system - Pathology 16006
Sense organs - Physiology and biochemistry 20004
Pharmacology - Clinical pharmacology 22005
Pharmacology - Immunological processes and allergy 22018
Immunology - General and methods 34502

Chemotherapy - General, methods and metabolism 38502
Chemotherapy - Antibacterial agents 38504
Plant physiology - Respiration, fermentation 51508
Pharmacognosy and pharmaceutical botany 54000

INDEX TERMS: Major Concepts
Immune System (Chemical Coordination and Homeostasis); Pharmacognosy (Pharmacology); Respiratory System (Respiration); Sense Organs (**Sensory** Reception)

INDEX TERMS: Parts, Structures, & Systems of Organisms
nasal **epithelium**: **respiratory** system, **sensory** system

INDEX TERMS: Diseases
upper airway disease: respiratory system disease, drug therapy

INDEX TERMS: Chemicals & Biochemicals
Pelargonium sidoides root extract: antibacterial-drug, antiinfective-drug, immunologic-drug

INDEX TERMS: Miscellaneous Descriptors
ciliary beat frequency; **pharmacological** effect

ORGANISM: Classifier
Geraniaceae 26105
Super Taxa
Dicotyledones; Angiospermae; Spermatophyta; Plantae
Organism Name
Pelargonium sidoides (species)
Taxa Notes
Angiosperms, Dicots, Plants, Spermatophytes, Vascular Plants

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human (common)
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L136 ANSWER 42 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:109534 BIOSIS
DOCUMENT NUMBER: PREV200100109534
TITLE: Neurogenic influence in inflammatory sensitivity to airway pollutants.
AUTHOR(S): Roy, J. [Reprint author]; Oortgiesen, M.; Carter, J. D.; Boyes, W. K.; Veronesi, B.
CORPORATE SOURCE: National Research Council, Washington, DC, USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-719.11. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.
ISSN: 0190-5295.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Feb 2001
Last Updated on STN: 15 Feb 2002

Searched by Barb O'Bryen, STIC 2-2518

ABSTRACT:Particulate matter (PM) is a major **air pollutant** that has been associated with respiratory disorders in susceptible populations. Previous work has demonstrated that PM activates **sensory** receptors (i.e. capsaicin, pH sensitive) found on nerve fibers that innervate the airways and on bronchial epithelial cells. Activation of these receptors initiates a process known as neurogenic inflammation which results in neuropeptide and cytokine release. Previous experiments report that the BALB/c mouse strain is responsive to PM-inflammation in contrast to the non-responsive C57/blk (B6) mouse strain. This differential sensitivity is retained in **sensory** neurons **cultured** from dorsal root ganglia (DRG) and trigeminal ganglia (TG), since neurons from BALB/c **sensory** ganglia release higher levels of inflammatory cytokines in response to PM and other irritants (acid pH, capsaicin) relative to B6 neurons. In the present study, we use RT-PCR, cobalt histochemistry and immnocytochemical techniques to show that the expression of capsaicin (VR-1) and Substance P (NK-1) receptors and release of inflammatory cytokines and neuropeptides are higher in **sensory** neurons from BALB/c strain relative to B6. In conclusion, strain-specific inflammation to PM and other irritants appears to be mediated by the population of irritant and neuropeptide receptors found on DRG and TG neurons. J. Roy holds a National Research Council research associate award at the National Health and Environmental Effects Research Laboratory, N.C.

CONCEPT CODE:
Cytology - Animal 02506
General biology - Symposia, transactions and proceedings 00520
Biochemistry studies - Nucleic acids, purines and pyrimidines 10062
Biochemistry studies - Proteins, peptides and amino acids 10064
Respiratory system - Physiology and biochemistry 16004
Respiratory system - Pathology 16006
Nervous system - Physiology and biochemistry 20504
Nervous system - Pathology 20506
Immunology - Immunopathology, tissue immunology 34508

INDEX TERMS:
Major Concepts
Nervous System (Neural Coordination); Respiratory System (Respiration)

INDEX TERMS:
Parts, Structures, & Systems of Organisms
airway: respiratory system; bronchial **epithelial** cells: **respiratory** system; dorsal root ganglia: nervous system; **sensory** neurons: nervous system; trigeminal ganglia: nervous system

INDEX TERMS:
Diseases
neurogenic inflammation: immune system disease, nervous system disease

INDEX TERMS:
Diseases
respiratory disorder: respiratory system disease

INDEX TERMS:
Chemicals & Biochemicals
capsaicin receptors [VR-1 receptor]: expression; inflammatory cytokines; neuropeptides; particulate matter: **air pollutant**, inflammatory sensitivity; substance P, receptor [NK-1 receptor]: expression

INDEX TERMS:
Methods & Equipment
cobalt histochemistry: analytical method; immunocytochemical techniques: analytical method; reverse transcription-polymerase chain reaction: analytical method

INDEX TERMS:
Miscellaneous Descriptors
Meeting Abstract

ORGANISM:
Classifier

Searched by Barb O'Bryen, STIC 2-2518

Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse: BALB/c, C57/B6
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

L136 ANSWER 43 OF 47 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1977:168973 BIOSIS
DOCUMENT NUMBER: PREV197763063837; BA63:63837
TITLE: STUDIES ON CYSTIC FIBROSIS USING ISO ELECTRIC FOCUSING PART 3 CORRELATION BETWEEN CYSTIC FIBROSIS PROTEIN AND CILIARY DYS KINESIA ACTIVITY IN SERUM SHOWN BY A MODIFIED RABBIT TRACHEAL **BIOASSAY.**
AUTHOR(S): WILSON G B; MOSHER M T; FUDENBERG H H
SOURCE: Pediatric Research, (1977) Vol. 11, No. 2, pp. 143-146. CODEN: PEREBL. ISSN: 0031-3998.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ABSTRACT:A modified rabbit tracheal **bioassay** was developed for use in investigating a possible correlation between cystic fibrosis protein (CFP) and ciliary dyskinesia factor (CDF) in human serum. The **bioassay** requires high standards of tissue selection, and all epithelial tissue must be free of underlying connective tissue. When serum **samples** were collected and processed carefully and warmed to 37° before assay, CDF could be reliably detected in 31 of 31 sera from cystic fibrosis (CF) homozygotes or obligate heterozygotes in 35 min or less without prior fractionation or connection of sera, whereas 13 of 14 normal control sera were nonreactive. CDF-positive serum reacts in 3 consecutive phases: initial increase in **ciliary beat** frequency, **ciliary** dyskinesia and tissue destruction with extrusion of single ciliated cells, mucus and debris. These results confirm the association of CDF with cystic fibrosis. The **bioassay** is not specific for CF when whole sera are ***bioassayed*** , since serum from several patients with bronchial asthma also caused ciliary dyskinesia. This finding need not preclude using rabbit tracheal **ciliated epithelial** tissue as an assay for following the purification of CDF. Isoelectric focusing showed that the presence or absence of CFP corresponded with that of dyskinesia activity in all sera tested except for the active **samples** from 7 asthma patients, which were negative for CFP. CFP and CDF may be identical or closely related markers for the CF gene. The activity detected by the rabbit tracheal ***bioassay*** in sera from patients with asthma and other diseases probably is caused by a substance different from a CF-specific CDF. Several substances in human serum can produce ciliary dyskinesia in rabbit tracheal epithelium, but only 1 appears to be a specific marker for CF. CFP may be related structurally or metabolically to a CF-specific CDF. Patients with bronchial asthma and other respiratory and autoimmune disorders may harbor a substance that can produce ciliary dyskinesia but that differs from CF-specific CDF.
CONCEPT CODE: Genetics - Human 03508
Clinical biochemistry - General methods and applications 10006
Biochemistry methods - Proteins, peptides and amino acids 10054
Biochemistry studies - Proteins, peptides and amino acids 10064
Biophysics - Methods and techniques 10504

Searched by Barb O'Bryen, STIC 2-2518

Movement 12100
Pathology - Inflammation and inflammatory disease 12508
Metabolism - Metabolic disorders 13020
Blood - Blood and lymph studies 15002
Respiratory system - Pathology 16006
Development and Embryology - Pathology 25503
In vitro cellular and subcellular studies 32600
Immunology - Immunopathology, tissue immunology 34508
Allergy 35500

INDEX TERMS: Major Concepts
Biochemistry and Molecular Biophysics; Blood and Lymphatics (Transport and Circulation); Clinical Chemistry (Allied Medical Sciences); Genetics; Metabolism; Pulmonary Medicine (Human Medicine, Medical Sciences)

INDEX TERMS: Miscellaneous Descriptors
HUMAN BRONCHIAL ASTHMA

ORGANISM: Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates

ORGANISM: Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates

L136 ANSWER 44 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2000:8235 TOXCENTER
DOCUMENT NUMBER: PubMed ID: 10564181
TITLE: Effects of aqueous extracts of PM(10) filters from the Utah valley on human airway epithelial cells
AUTHOR(S): Frampton M W; Ghio A J; Samet J M; Carson J L; Carter J D; Devlin R B
CORPORATE SOURCE: Department of Medicine, University of Rochester School of Medicine and Dentistry, Rochester, New York 14642, USA
CONTRACT NUMBER: RO1-ES-02679 (NIEHS)
RO1-HL-51701 (NHLBI)
SOURCE: American journal of physiology, (1999 Nov) 277 (5 Pt 1) L960-7.
Journal Code: 0370511. ISSN: 0002-9513.
COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: MEDLINE
OTHER SOURCE: MEDLINE 2000035097
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20011116

ABSTRACT:
We hypothesized that the reduction in hospital respiratory admissions in the Utah Valley during closure of a local steel mill in 1986-1987 was attributable in part to decreased toxicity of ambient air particles. **Sampling** filters for particulate matter < 10 micrometer (PM(10)) were obtained from a Utah Valley monitoring station for the year before (year 1), during (year 2), and after (year 3) the steel mill closure. Aqueous extracts of the filters

were analyzed for metal content and oxidant production and added to ***cultures*** of human **respiratory epithelial** (BEAS-2B) cells for 2 or 24 h. Year 2 dust contained the lowest concentrations of soluble iron, copper, and zinc and showed the least oxidant generation. Only dust from year 3 caused cytotoxicity (by microscopy and lactate dehydrogenase release) at 500 microgram/ml. Year 1 and year 3, but not year 2, dust induced expression of interleukin-6 and -8 in a dose-response fashion. The effects of ambient air particles on human respiratory epithelial cells vary significantly with time and metal concentrations.

CONTROLLED TERM: Check Tags: Comparative Study
*Air Pollutants, Occupational: TO, toxicity
Cytotoxins: PD, pharmacology
*Epithelial Cells: DE, drug effects
Epithelial Cells: IM, immunology
Epithelial Cells: UL, ultrastructure
Filtration
Gene Expression: DE, drug effects
Gene Expression: IM, immunology
Humans
Interleukin-6: GE, genetics
Interleukin-6: IM, immunology
Interleukin-8: GE, genetics
Interleukin-8: IM, immunology
*Lung: CY, cytology
Metals, Heavy: PD, pharmacology
Microscopy, Electron, Scanning
Occupational Diseases: CI, chemically induced
Occupational Diseases: IM, immunology
Occupational Diseases: PA, pathology
Oxidative Stress: DE, drug effects
RNA, Messenger: AN, analysis
Research Support, U.S. Gov't, P.H.S.
*Steel
Utah
Ventilation
Water

REGISTRY NUMBER: 12597-69-2 (Steel)
7732-18-5 (Water)

CHEMICAL NAME: 0 (**Air Pollutants**, Occupational); 0 (Cytotoxins); 0 (Interleukin-6); 0 (Interleukin-8); 0 (Metals, Heavy); 0 (RNA, Messenger)

L136 ANSWER 45 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:325342 TOXCENTER
DOCUMENT NUMBER: CRISP-2003-ES009844-04
TITLE: **SENSORY** IRRITANT RECEPTORS AND PARTICULATE INFLAMMATION

AUTHOR(S): SIMON S A
CORPORATE SOURCE: SAS@NEURO.DUKE.EDU, DUKE UNIVERSITY MEDICAL CENTER, BOX 3209, DURHAM, NC 27710:NORTH CAROLINA

SUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES

SOURCE: Crisp Data Base National Institutes of Health.
DOCUMENT TYPE: (Research)
FILE SEGMENT: CRISP
LANGUAGE: English
ENTRY DATE: Entered STN: 20041229

Searched by Barb O'Bryen, STIC 2-2518

Last Updated on STN: 20041229

ABSTRACT:
Increases in pulmonary related incidents of morbidity and mortality have been epidemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM toxicity are poorly understood. This proposal examines the hypothesis that physicochemical components of PM initiate inflammation by activation of irritant (i.e., capsaicin, acid-sensitive) receptors located on **sensory** nerve endings and airway epithelial target cells. This activation results in an influx of extracellular calcium and release of neuropeptides and inflammatory cytokines, which proceed to initiate and sustain events of airway inflammation ands hypersensitivity. The response of irritant receptors to PM will be examined in primary **cultures** of **sensory** dorsal root ganglion neurons and in human bronchial epiothelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cytokine release) endpoints will be used to characterize the response to various urban, industrial and ambient PM. The physicochemical properties of selected PM and their separate soluble and particulate fractions will be determined and examined for their cellular effects. Based on these data,. synthetic particle analogues will be designed, that resemble PM particles in size and surface charge, to assess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characterize the contribution of culpable irritant receptors to the PM-mediated effects. In addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined in this proposal will identify the responsible factors and target sites contributing to PM toxicity.
SUPPLEMENTARY TERMS: Miscellaneous Descriptors
air pollution; laboratory mouse; inflammation; irritation ,irritant; pharmacokinetics; environmental health; environmental contamination; spinal ganglion; particle; **respiratory epithelium**; tissue ,cell **culture**; environmental toxicology; **sensory** receptor

L136 ANSWER 46 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:156403 TOXCENTER
DOCUMENT NUMBER: CRISP-2002-ES09844-03
TITLE: **SENSORY** IRRITANT RECEPTORS AND PARTICULATE INFLAMMATION
AUTHOR(S): SIMON S A
CORPORATE SOURCE: SAS@NEURO.DUKE.EDU,. DUKE UNIVERSITY MEDICAL CENTER, BOX 3209, DURHAM, NC 27710:NORTH CAROLINA
SUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES
SOURCE: Crisp Data Base National Institutes of Health.
DOCUMENT TYPE: (Research)
FILE SEGMENT: CRISP
LANGUAGE: English
ENTRY DATE: Entered STN: 20030708
Last Updated on STN: 20030708

ABSTRACT:
Increases in pulmonary related incidents of morbidity and mortality have been epidemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM toxicity are poorly understood. This proposal examines the hypothesis that physicochemical components of PM initiate inflammation by activation of irritant (i.e., capsaicin, acid-sensitive) receptors located on **sensory**

nerve endings and airway epithelial target cells. This activation results in an influx of extracellular calcium and release of neuropeptides and inflammatory cytokines, which proceed to initiate and sustain events of airway inflammation ands hypersensitivity. The response of irritant receptors to PM will be examined in primary **cultures** of **sensory** dorsal root ganglion neurons and in human bronchial epiothelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cytokine release) endpoints will be used to characterize the response to various urban, industrial and ambient PM. The physicochemical properties of selected PM and their separate soluble and particulate fractions will be determined and examined for their cellular effects. Based on these data, synthetic particle analogues will be designed, that resemble PM particles in size and surface charge, to assess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characterize the contribution of culpable irritant receptors to the PM-mediated effects. In addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined in this proposal will identify the responsible factors and target sites contributing to PM toxicity.

SUPPLEMENTARY TERMS: Miscellaneous Descriptors
air pollution; laboratory mouse; inflammation; irritation ,irritant; pharmacokinetics; environmental health; environmental contamination; spinal ganglion; particle; **respiratory epithelium**; tissue ,cell **culture**; environmental toxicology; **sensory** receptor

L136 ANSWER 47 OF 47 TOXCENTER COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:559667 TOXCENTER
DOCUMENT NUMBER: CRISP-2000-ES09844-01A2
TITLE: **SENSORY** IRRITANT RECEPTORS AND PARTICULATE INFLAMMATION
AUTHOR(S): SIMON S A
CORPORATE SOURCE: DUKE UNIVERSITY MEDICAL CENTER, BOX 3209, DURHAM, NC 27710:NORTH CAROLINA
SUPPORTING ORGANIZATION (SPONSORING AGENCY): U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE OF ENVIRONMENTAL HEALTH SCIENCES
SOURCE: Crisp Data Base National Institutes of Health.
DOCUMENT TYPE: (Research)
FILE SEGMENT: CRISP
LANGUAGE: English
ENTRY DATE: Entered STN: 20021200
Last Updated on STN: 20021200

ABSTRACT:
Increases in pulmonary related incidents of morbidity and mortality have been ep idemiologically associated with exposure to particulate matter (PM). Due to its heterogeneity and complex physicochemistry, the underlying mechanism(s) of PM t oxicity are poorly understood. This proposal examines the hypothesis that physi cochemical components of PM initiate inflammation by activation of irritant (i.e ., capsaicin, acid-sensitive) receptors located on ***sensory*** nerve endings and air way epithelial target cells. This activation results in an influx of extracellu lar calcium and release of neuropeptides and inflammatory cytokines, which proce ed to initiate and sustain events of airway inflammation ands hypersensitivity. The response of irritant receptors to PM will be examined in primary **cultures** o f ***sensory*** dorsal root ganglion neurons and in human bronchial epiothelial cells. Biophysical (e.g., patch clamp, calcium imaging) and immunological (e.g., cyto kine release) endpoints will be used to characterize the response

to various urb an, industrial and ambient PM. The physicochemical properties of selected PM an d their separate soluble and particulate fractions will be determined and examin ed for their cellular effects. Based on these data, synthetic particle analogue s will be designed, that resemble PM particles in size and surface charge, to as sess the inflammatory contribution of specific PM components. Physiological and pharmacological evaluation of the responses to PM will identify and characteriz e the contribution of culpable irritant receptors to the PM-mediated effects. I n addition, the involvement of other cellular pathways (i.e., calcium channels, acid-sensitive exchangers) will be examined. The mechanistic approach outlined i n this proposal will identify the responsible factors and target sites contribut ing to PM toxicity.

SUPPLEMENTARY TERMS: Miscellaneous Descriptors
air pollution; laboratory mouse;
inflammation; irritation ,irritant; pharmacokinetics;
environmental health; environmental contamination; spinal
ganglion; particle; **respiratory**
epithelium; tissue ,cell **culture**;
environmental toxicology; **sensory** receptor

FILE 'HOME' ENTERED AT 16:11:16 ON 16 DEC 2005

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Searched by Barb O'Bryen, STIC 2-2518

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=> d his nofile

     (FILE 'HOME' ENTERED AT 14:50:08 ON 16 DEC 2005)

     FILE 'CAPLUS' ENTERED AT 14:50:30 ON 16 DEC 2005
               SET LINE 250
               SET DETAIL OFF
               E US2004-798986/AP,PRN 25
               SET LINE LOGIN
               SET DETAIL LOGIN
L1          493 SEA ABB=ON  CHIN W?/AU
L2         2192 SEA ABB=ON  KWON S?/AU
L3            0 SEA ABB=ON  L1 AND L2
               E BIOSENSOR/CT
               E E4+ALL
L4        16036 SEA ABB=ON  BIOSENSORS/CT
L5            2 SEA ABB=ON  (L1 OR L2) AND L4
               D SCAN
L6         3734 SEA ABB=ON  EPITHELI?/OBI(L)(RESPIRATORY/OBI OR CILIAT?/OBI OR
               GOBLET?/OBI)
L7            1 SEA ABB=ON  L4 AND L6
               D SCAN
               E ENVIRONMENTAL POLLUTION/CT
               E E3+ALL
L8       208880 SEA ABB=ON  CULTUR?/OBI
L9          244 SEA ABB=ON  L6 AND L8
L10       42474 SEA ABB=ON  ENVIRONMENTAL POLLUTION/CT
L11      147315 SEA ABB=ON  AIR/OBI (L) POLLUTION/CW
L12        4705 SEA ABB=ON  (BIOLOGICAL/OBI OR CHEMICAL/OBI)(L)WARFARE/OBI
L13       13214 SEA ABB=ON  AIR/OBI(L)MONITOR?/OBI
L14       11081 SEA ABB=ON  SAMPLING/CT
L15           4 SEA ABB=ON  L9 AND (L10 OR L11 OR L12 OR L13 OR L14)
               D SCAN.
L16       11248 SEA ABB=ON  AIRBORNE PARTICLES/CT
L17       56518 SEA ABB=ON  TOXICITY/CT
L18        4102 SEA ABB=ON  ECOTOXICITY/CT
L19        9069 SEA ABB=ON  BIOASSAY/CT
L20      427058 SEA ABB=ON  59/SC,SX
L21      615834 SEA ABB=ON  4/SC,SX
L22          14 SEA ABB=ON  L6 AND L19
L23           3 SEA ABB=ON  L22 AND ((L10 OR L11 OR L12 OR L13 OR L14) OR (L16
               OR L17 OR L18))
L24         124 SEA ABB=ON  L8 AND L20 AND L21
L25          74 SEA ABB=ON  L8 AND L20 AND L21 AND ((L10 OR L11 OR L12 OR L13)
               OR L16)
L26       20819 SEA ABB=ON  SAMPLING/CW
L27           4 SEA ABB=ON  L9 AND ((L10 OR L11 OR L12 OR L13) OR L26)
L28           3 SEA ABB=ON  L22 AND ((L10 OR L11 OR L12 OR L13) OR L26 OR (L16
               OR L17 OR L18))
L29           3 SEA ABB=ON  L25 AND L26
L30           3 SEA ABB=ON  L29 NOT (L7 OR L27 OR L28)
               D SCAN TI
L31         647 SEA ABB=ON  (CILIA?(2A)BEAT?)/BI
L32        2600 SEA ABB=ON  (ELECTRICAL RESPONS?)/BI
L33        1144 SEA ABB=ON  (SECRET?(2A)MUCIN#)/BI
L34          28 SEA ABB=ON  L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11
               OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20 OR L21) OR
               L26)
               D QUE
L35           9 SEA ABB=ON  L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11
```

OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20) OR L26)
D SCAN TI

L36 3 SEA ABB=ON VITRO/TI AND L35
D SCAN

L37 8 SEA ABB=ON L6 AND (L31 OR L32 OR L33) AND (L4 OR (L10 OR L11 OR L12 OR L13) OR (L16 OR L17 OR L18 OR L19 OR L20) OR L26) AND L21

INDEX '1MOBILITY, 2MOBILITY, ABI-INFORM, ADISCTI, AEROSPACE, AGRICOLA, ALUMINIUM, ANABSTR, ANTE, APOLLIT, AQUALINE, AQUASCI, AQUIRE, BABS, BIBLIODATA, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, BLLDB, CABA, CANCERLIT, ...' ENTERED AT 15:10:49 ON 16 DEC 2005

SEA (AIR(2A)(POLLUTION OR QUALITY)) OR AIRBORNE OR ((BIOLOGICAL

2357	FILE 1MOBILITY
81	FILE 2MOBILITY
13485	FILE ABI-INFORM
298	FILE ADISCTI
51582	FILE AEROSPACE
12003	FILE AGRICOLA
931	FILE ALUMINIUM
2212	FILE ANABSTR
2308	FILE ANTE
106	FILE APOLLIT
2064	FILE AQUALINE
5982	FILE AQUASCI
19	FILE AQUIRE
1154	FILE BABS
256	FILE BIBLIODATA
16668	FILE BIOBUSINESS
143	FILE BIOCOMMERCE
3018	FILE BIOENG
45230	FILE BIOSIS
299	FILE BIOTECHABS
299	FILE BIOTECHDS
4090	FILE BIOTECHNO
26656	FILE CABA
2070	FILE CANCERLIT
1005	FILE CAOLD
171800	FILE CAPLUS
88	FILE CASREACT
2471	FILE CBNB
6473	FILE CEABA-VTB
898	FILE CEN
798	FILE CERAB
4901	FILE CIN
6092	FILE CIVILENG
72364	FILE COMPENDEX
1817	FILE COMPUAB
224	FILE COMPUSCIENCE
4698	FILE CONFSCI
575	FILE COPPERLIT
310	FILE CORROSION
136	FILE CROPB
394	FILE CROPU
2869	FILE CSNB
132	FILE DDFB
67	FILE DDFU
2	FILE DETHERM

3354	FILE DGENE
3491	FILE DISSABS
199	FILE DKF
2083	FILE DPCI
132	FILE DRUGB
122	FILE DRUGU
1765	FILE ELCOM
279	FILE EMA
270	FILE EMBAL
53194	FILE EMBASE
46972	FILE ENCOMPLIT
7058	FILE ENCOMPPAT
157337	FILE ENERGY
20627	FILE ENTEC
15699	FILE ENVIROENG
6514	FILE EPFULL
11375	FILE ESBIOBASE
1	FILE FOMAD
7	FILE FORIS
134	FILE FRANCEPAT
529	FILE FRFULL
656	FILE FROSTI
753	FILE FSTA
2691	FILE GBFULL
705	FILE GENBANK
19183	FILE GEOREF
8597	FILE HEALSAFE
5329	FILE ICONDA
3	FILE IFICLS
7721	FILE IFIPAT
12	FILE IMSDRUGNEWS
23	FILE INFODATA
21605	FILE INIS
5150	FILE INPADOC
46245	FILE INSPEC
1726	FILE INSPHYS
31300	FILE INVESTEXT
409	FILE IPA
8635	FILE ITRD
1753	FILE JAPIO
27982	FILE JICST-EPLUS
613	FILE KOREAPAT
116	FILE KOSMET
10472	FILE LIFESCI
96	FILE LISA
920	FILE MATBUS
425	FILE MATH
12356	FILE MECHENG
35790	FILE MEDLINE
2588	FILE METADEX
14	FILE NAPRALERT
16557	FILE NIOSHTIC
46175	FILE NLDB
82731	FILE NTIS
4	FILE NUTRACEUT
2630	FILE OCEAN
4669	FILE PAPERCHEM2
78743	FILE PASCAL
4	FILE PATDPA
246	FILE PATDPAFULL

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      11213  FILE PCTFULL
         18  FILE PHARMAML
        326  FILE PHIN
       2542  FILE PIRA
      38053  FILE POLLUAB
      76931  FILE PROMT
       2251  FILE RAPRA
       5643  FILE RSWB
        287  FILE RUSSIAPAT
      39834  FILE SCISEARCH
        539  FILE SOLIDSTATE
        203  FILE SOLIS
      10988  FILE TEMA
       2481  FILE TEXTILETECH
     149730  FILE TOXCENTER
         73  FILE TRIBO
       7341  FILE TULSA
       6609  FILE TULSA2
       4561  FILE UFORDAT
      36533  FILE ULIDAT
      46618  FILE USPATFULL
       3917  FILE USPAT2
         23  FILE VETB
         42  FILE VETU
       8695  FILE WATER
        438  FILE WELDASEARCH
      12737  FILE WPIDS
         55  FILE WPIFV
      12737  FILE WPINDEX
       2497  FILE WSCA
        906  FILE WTEXTILES
L38           QUE ABB=ON  (AIR(2A)(POLLUTION OR QUALITY)) OR AIRBORNE OR
              ((BIOLOGICAL OR CHEMICAL)(2A) WARFARE)
              ---------
              D RANK

     FILE 'STNGUIDE' ENTERED AT 15:14:17 ON 16 DEC 2005
L39           0 SEA ABB=ON  CHIN W?/AU
L40           0 SEA ABB=ON  KWON S?/AU
L41           0 SEA ABB=ON  BIOSENSOR# OR SENSOR#
L42           0 SEA ABB=ON  BIOASSAY?
L43           3 SEA ABB=ON  CULTUR?
L44          13 SEA ABB=ON  (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR
                 QUALITY)
L45           0 SEA ABB=ON  AIRBORNE OR AIR BORNE
L46           0 SEA ABB=ON  (BIOLOGICAL OR CHEMICAL)(2A)WARFARE
L47           0 SEA ABB=ON  (CILIA?(2A)BEAT?)
L48           0 SEA ABB=ON  (ELECTRICAL RESPONS?)
L49           0 SEA ABB=ON  (SECRET?(2A)MUCIN#)

     FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIOBASE, NTIS, NIOSHTIC,
     ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
     TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 15:23:02 ON 16 DEC 2005
L50        3121 SEA ABB=ON  L1
L51        7974 SEA ABB=ON  L2
L52     2197473 SEA ABB=ON  BIOSENSOR# OR SENSOR#
L53      290424 SEA ABB=ON  BIOASSAY?
L54       29232 SEA ABB=ON  EPITHELI?(3A)(RESPIRATORY OR CILIAT? OR GOBLET?)
L55     3624943 SEA ABB=ON  CULTUR?
L56      955407 SEA ABB=ON  (ENVIRONMENT? OR AIR)(2A)(POLLUT? OR MONITOR? OR
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            QUALITY)
L57      158030 SEA ABB=ON  AIRBORNE OR AIR BORNE
L58       17285 SEA ABB=ON  (BIOLOGICAL OR CHEMICAL)(2A) WARFARE
L59        4285 SEA ABB=ON  (CILIA?(2A) BEAT?)
L60        7049 SEA ABB=ON  (ELECTRICAL RESPONS?)
L61        4997 SEA ABB=ON  (SECRET?(2A) MUCIN#)
L62           2 SEA ABB=ON  L50 AND L51
                D SCAN
L63           0 SEA ABB=ON  (L50 OR L51) AND (L52 OR L53) AND (L54 OR (L56 OR
                L57 OR L58))
                D KWIC L62
L64        1378 SEA ABB=ON  (L52 OR L53) AND L54
L65         122 SEA ABB=ON  (L52 OR L53) AND L54 AND L55
L66          16 SEA ABB=ON  L65 AND (L56 OR L57 OR L58)
L67           2 SEA ABB=ON  L54(5A) L55 AND (L56 OR L57 OR L58) AND (L59 OR
                L60 OR L61)
L68           1 SEA ABB=ON  L64 AND (L56 OR L57 OR L58) AND (L59 OR L60 OR
                L61)
L69          32 SEA ABB=ON  L64 AND (L59 OR L60 OR L61)
L70          25 DUP REM L69 (7 DUPLICATES REMOVED)
                    ANSWERS '1-3' FROM FILE JICST-EPLUS
                    ANSWERS '4-6' FROM FILE PASCAL
                    ANSWER '7' FROM FILE BIOTECHNO
                    ANSWERS '8-9' FROM FILE ESBIOBASE
                    ANSWERS '10-11' FROM FILE INSPEC
                    ANSWERS '12-23' FROM FILE BIOSIS
                    ANSWERS '24-25' FROM FILE SCISEARCH
                D SCAN
L71     1500078 SEA ABB=ON  TOXICITY/CT OR PHARMACOLOGICAL OR SCREEN?/TI
L72     4696113 SEA ABB=ON  SAMPL?
L73           9 SEA ABB=ON  L69 AND (L71 OR L72)
L74           6 SEA ABB=ON  L54(5A) L55 AND L72 AND (L56 OR L57 OR L58)
                D SCAN
L75       36347 SEA ABB=ON  UTAH
L76           4 SEA ABB=ON  L74 AND L75
                D KWIC

FILE 'MEDLINE' ENTERED AT 15:38:26 ON 16 DEC 2005
L77         447 SEA ABB=ON  CHIN W?/AU
L78         540 SEA ABB=ON  KWON S?/AU
L79           0 SEA ABB=ON  L77 AND L78

FILE 'STNGUIDE' ENTERED AT 15:38:40 ON 16 DEC 2005

FILE 'MEDLINE' ENTERED AT 15:39:31 ON 16 DEC 2005
L80        3896 SEA ABB=ON  BIOLOGICAL WARFARE+NT/CT
L81         716 SEA ABB=ON  CHEMICAL WARFARE+NT/CT

FILE 'STNGUIDE' ENTERED AT 15:40:38 ON 16 DEC 2005

FILE 'MEDLINE' ENTERED AT 15:40:46 ON 16 DEC 2005
L82      102328 SEA ABB=ON  ENVIRONMENTAL EXPOSURE+NT/CT
L83       41512 SEA ABB=ON  AIR POLLUTION+NT/CT

FILE 'STNGUIDE' ENTERED AT 15:42:22 ON 16 DEC 2005

FILE 'MEDLINE' ENTERED AT 15:42:30 ON 16 DEC 2005
L84      176076 SEA ABB=ON  EPITHELIAL CELLS+NT/CT
L85      174367 SEA ABB=ON  EPITHELIUM+NT/CT
L86      269379 SEA ABB=ON  RESPIRATORY SYSTEM+NT/CT
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FILE 'STNGUIDE' ENTERED AT 15:43:47 ON 16 DEC 2005

FILE 'MEDLINE' ENTERED AT 15:43:57 ON 16 DEC 2005

L87 7060 SEA ABB=ON CILIA/CT
L88 485 SEA ABB=ON GOBLET CELLS+NT/CT
L89 10594 SEA ABB=ON MUCINS+NT/CT
L90 8627 SEA ABB=ON BIOSENSING TECHNIQUES+NT/CT
L91 6517 SEA ABB=ON BIOSENSORS/CT
L92 0 SEA ABB=ON L91 NOT L90
L93 758406 SEA ABB=ON CELLS, CULTURED+NT/CT
L94 13 SEA ABB=ON (L77 OR L78) AND (L80 OR L81 OR L82 OR L83)
L95 1 SEA ABB=ON (L77 OR L78) AND (L80 OR L81 OR L82 OR L83) AND ((L84 OR L85 OR L86 OR L87 OR L88 OR L89 OR L90) OR L93)
D SCAN
D TRIAL
L96 437 SEA ABB=ON (L80 OR L81 OR L82 OR L83) AND ((L84 OR L85) AND L86)
L97 129 SEA ABB=ON L96 AND L93
L98 0 SEA ABB=ON L97 AND (L90 OR L91)
L99 5 SEA ABB=ON L97 AND (L87 OR L88 OR L89)
D TRIAL 1-5
E AIR POLLUTANTS, ENVIRONMENTAL+ALL/CT
L100 60664 SEA ABB=ON AIR POLLUTANTS+NT/CT
D QUE L99
L101 9 SEA ABB=ON ((L80 OR L81 OR L82 OR L83) OR L100) AND (L84 OR L85) AND L86 AND L93 AND (L87 OR L88 OR L89)

FILE 'EMBASE' ENTERED AT 15:51:55 ON 16 DEC 2005

L102 395 SEA ABB=ON CHIN W?/AU
L103 476 SEA ABB=ON KWON S?/AU
D QUE L101
E BIOLOGICAL WARFARE+ALL/CT
L104 3037 SEA ABB=ON BIOLOGICAL WARFARE/CT
E CHEMICAL WARFARE+ALL/CT
L105 996 SEA ABB=ON CHEMICAL WARFARE/CT
E ENVIRONMENTAL EXPOSURE+ALL/CT
L106 22836 SEA ABB=ON ENVIRONMENTAL EXPOSURE/CT
E AIR POLLUTION+ALL/CT
L107 84207 SEA ABB=ON "AIR AND AIR RELATED PHENOMENA"+NT/CT
D QUE L101
E AIR POLLUTANTS+ALL
E AIR POLLUTANTS+ALL/CT
E E2+ALL
D QUE L101
E RESPIRATORY EPITH/CT
E E7+ALL
L108 3900 SEA ABB=ON RESPIRATORY EPITHELIUM/CT
E CELLS, CULTURED+ALL/CT
L109 187739 SEA ABB=ON CELL CULTURE/CT
E CILIA/CT
E E3+ALL
L110 218 SEA ABB=ON EUKARYOTIC FLAGELLUM/CT
E RESPIRATORY TRACT CIL/CT
E GOBLET CELLS/CT
E E3+ALL
L111 2385 SEA ABB=ON GOBLET CELL/CT
E MUCIN+ALL/CT
L112 6530 SEA ABB=ON MUCIN/CT
D QUE L101

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              D QUE L67
              D QUE L68
              D QUE L73
              D QUE L74
L128          0 S (L66-L68 OR L73-L74) NOT L62

     FILE 'EMBASE' ENTERED AT 16:08:34 ON 16 DEC 2005
              D QUE L118
              D QUE L123
              D QUE L126
L128          6 SEA ABB=ON  L118 OR L123 OR L126

     FILE 'CAPLUS' ENTERED AT 16:08:36 ON 16 DEC 2005
              D QUE L7
              D QUE L27
              D QUE L28
              D QUE L30
              D QUE L37
L129         18 SEA ABB=ON  (L7 OR L27 OR L28 OR L30 OR L37) NOT L5

     FILE 'MEDLINE' ENTERED AT 16:08:38 ON 16 DEC 2005
              D QUE L101
L130          9 SEA ABB=ON  L101 NOT L95

     FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIOBASE, NTIS, NIOSHTIC,
     ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
     TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:08:50 ON 16 DEC 2005
              D QUE L66
              D QUE L67
              D QUE L68
              D QUE L73
              D QUE L74
L131         19 SEA ABB=ON  (L66 OR L67 OR L68)
L132         15 SEA ABB=ON  (L73 OR L74)
L133         19 SEA ABB=ON  L131 NOT L62
L134         15 SEA ABB=ON  L132 NOT L62
L135         34 SEA ABB=ON  (L131 OR L132)
     FILE 'STNGUIDE' ENTERED AT 16:10:18 ON 16 DEC 2005

     FILE 'MEDLINE, CAPLUS, EMBASE, PASCAL, ESBIOBASE, NIOSHTIC, INSPEC,
     BIOSIS, LIFESCI, TOXCENTER, SCISEARCH' ENTERED AT 16:10:42 ON 16 DEC 2005
L136         47 DUP REM L130 L129 L128 L135 (20 DUPLICATES REMOVED)
                ANSWERS '1-9' FROM FILE MEDLINE
                ANSWERS '10-26' FROM FILE CAPLUS
                ANSWERS '27-31' FROM FILE EMBASE
                ANSWERS '32-33' FROM FILE PASCAL
                ANSWERS '34-36' FROM FILE ESBIOBASE
                ANSWER '37' FROM FILE NIOSHTIC
                ANSWER '38' FROM FILE INSPEC
                ANSWERS '39-43' FROM FILE BIOSIS
                ANSWERS '44-47' FROM FILE TOXCENTER
              D IALL 1-9
              D IBIB ED ABS HITIND 10-26
              D IALL 27-47

     FILE 'HOME' ENTERED AT 16:11:16 ON 16 DEC 2005

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                E BIOSENS/CT
                E E5+ALL
L113         7849 SEA ABB=ON  BIOSENSOR+NT/CT
                E BIOASSAY/CT
                E E3+ALL
L114        15116 SEA ABB=ON  BIOASSAY/CT
L115            0 SEA ABB=ON  L102 AND L103
L116            0 SEA ABB=ON  (L102 OR L103) AND (L104 OR L105 OR L106 OR L107)
                  AND (L108 OR L109 OR L110 OR L111 OR L112 OR L113 OR L114)
L117           12 SEA ABB=ON  (L104 OR L105 OR L106 OR L107) AND L108 AND L109
L118            1 SEA ABB=ON  (L104 OR L105 OR L106 OR L107) AND L108 AND (L113
                  OR L114)
                D TRIAL L117 1-12
L119       578914 SEA ABB=ON  IN VITRO STUDY/CT
L120            0 SEA ABB=ON  CELL MONOLAYER/CT
L121         4485 SEA ABB=ON  MONOLAYER CULTURE/CT
L122         1551 SEA ABB=ON  CILIARY MOTILITY/CT
L123            4 SEA ABB=ON  (L104 OR L105 OR L106 OR L107) AND L108 AND L109
                  AND (L119 OR (L121 OR L122))
L124            8 SEA ABB=ON  L117 NOT (L118 OR L123)
                D TRIAL 1-18
L125        38308 SEA ABB=ON  QUANTITATIVE ANALYSIS/CT
L126            1 SEA ABB=ON  L117 AND L125

FILE 'STNGUIDE' ENTERED AT 16:04:24 ON 16 DEC 2005

FILE 'CAPLUS' ENTERED AT 16:04:57 ON 16 DEC 2005
                D QUE L5
                D QUE L3

FILE 'MEDLINE' ENTERED AT 16:04:57 ON 16 DEC 2005
                D QUE L95

FILE 'EMBASE' ENTERED AT 16:04:57 ON 16 DEC 2005
                D QUE L115
                D QUE L116.

FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIOBASE, NTIS, NIOSHTIC,
ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:05:30 ON 16 DEC 2005
                D QUE L62
                D QUE L63

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 16:05:42 ON 16 DEC
2005
L127            5 DUP REM L95 L5 L62 (0 DUPLICATES REMOVED)
                    ANSWER '1' FROM FILE MEDLINE
                    ANSWERS '2-3' FROM FILE CAPLUS
                    ANSWER '4' FROM FILE BIOSIS
                    ANSWER '5' FROM FILE SCISEARCH
                D IALL 1
                D IBIB ED ABS HITIND 2-3
                D IALL 4-5

FILE 'STNGUIDE' ENTERED AT 16:06:10 ON 16 DEC 2005

FILE 'JICST-EPLUS, PASCAL, CABA, BIOTECHNO, ESBIOBASE, NTIS, NIOSHTIC,
ENVIROENG, HEALSAFE, INSPEC, BIOSIS, CONFSCI, LIFESCI, POLLUAB,
TOXCENTER, CEABA-VTB, WPIX, SCISEARCH' ENTERED AT 16:08:29 ON 16 DEC 2005
                D QUE L66
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Searched by Barb O'Bryen, STIC 2-2518